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EVALUATION OF STEREOPSIS

A COMPARISON OF THE HOWARD-DOLMAN AND THE VERHOEFF TEST

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Increasing use of visual tests in the field of personnel selection,[1] special military needs and the rapid expansion of aviation have stimulated interest in technics which measure stereopsis. For several years the Howard-Dolman test of stereopsis[2] (not to be confused with the original Howard test[3]) has been used extensively, and more recently Verhoeff[4] has described another method. Verhoeff's test evaluates the acuity of stereopsis by means of a simple device which causes the estimate of relative depth to be based on binocular parallax, eliminates eye-hand coordination and difference in image size as aids in forming judgments and permits little opportunity (1 chance in 65,000) for passing the test by guessing.[4] The results obtained when a group of adolescents was given both these tests are discussed in this report.

METHOD

The entire student population of a boys' preparatory school was given a thorough ophthalmic examination,[5] and from this group of 720 boys 186 who fulfilled the following requirements were selected for the tests of stereoscopic ability: normal color vision, a normal external ocular condition, normal fundi, normal pupils, a visual acuity of 20/20 in each eye, less than 1.5 D. of hyperopia, less than 1.25 prism diopters of hyperphoria, less than 4 prism diopters of exophoria and less than 6 prism diopters of esophoria at 20 feet (6 meters); less than 6 prism diopters of esophoria and less than 8 prism diopters of exophoria at 16 inches (40 cm.) and less than 1 D. of astigmatism. Hyperphoria was measured with the Maddox rod; esophoria and exophoria at 20 feet with the Maddox rod and the cover test, and esophoria and exophoria at 16 inches by von Graefe's prism test and the cover test.

Each of these 186 boys was subsequently given the Howard-Dolman test according to the method commonly employed[2]; three successive readings were taken and recorded. The Verhoeff test[4] consists of three vertical black paper strips viewed against a translucent background: The subject is asked to state which of the strips is nearest, or farthest from, him. The test device is first held at a distance of 210 cm. from the subject and then moved closer to him until the distance at which his response is correct can be determined. Eight different relative positions of the strips can be obtained, and the distance at which the subject makes the correct response to all these positions is recorded.

TABLE 1.—*Distribution of Averages of the First Three Trial Settings of the Howard-Dolman Apparatus for 186 Subjects*

Average Setting (Howard-Dolman), Mm.	Number of Subjects
0-4	34
5-9	63
10-14	40
15-19	20
20-24	9
25-29	7
30-34	3
35-39	3
40-44	0
45-49	3
50-54	0
55-59	0
60-64	2
65-69	2
Total	186

RESULTS

In table 1 is shown the distribution of the averages of the first three trial settings of the Howard-Dolman apparatus; about 85 per cent of the subjects had an average score of less than

1. Kuhn, H. S.: Significance of Visual Defects in War Production Effort, J. A. M. A. **123**:1085-1087 (Dec. 25) 1943.

2. Armstrong, H. G.: Principles and Practice of Aviation Medicine, Baltimore, Williams & Wilkins Company, 1939, p. 77.

3. Howard, H. J.: A Test for the Judgment of Distance, Tr. Am. Ophth. Soc. **17**:195, 1919.

4. Verhoeff, F. H.: Quantitative Test for Acuity and Reliability of Binocular Stereopsis, Arch. Ophth. **28**:1000-1014 (Dec.) 1942.

5. Sloane, A. E., and Gallagher, J. R.: A Summary of Findings at the Eye Examination of Preparatory School Boys, Am. J. Ophth. **26**:1076-1083 (Oct.) 1943.

20 mm., and about 5 per cent had average scores greater than 34 mm.

The distribution of the averages of the eight ratings on the Verhoeff test is given in table 2.

TABLE 2.—*Distribution of Averages for 186 Subjects of Distances at Which the Eight Correct Responses to the Verhoeff Test Were Made*

Average Rating (Verhoeff), Cm.	Number of Subjects
50	1
60	0
70	7
80	1
90	1
100	3
110	3
120	7
130	11
140	14
150	11
160	26
170	34
180	28
190	17
200 or more	22
Total	186

About 82 per cent of the group made correct responses at an average distance of 140 cm. or more, and about 5 per cent were unable to give correct responses unless the test object was 90 cm. or less away. The correlation between the averages of the three Howard-Dolman settings and the averages of the first three Verhoeff ratings was only 0.438.

In evaluating any testing method it is proper to determine how much variation exists between each one of a series of trials. Coefficients of self reliability between each of the first three Verhoeff ratings and between each of the three ratings on the Howard-Dolman test are listed in table 3. A considerably higher coefficient of

TABLE 3.—*Coefficients of Self Reliability Between First and Second, Second and Third and First and Third Howard-Dolman Settings and Between Similarly Related Verhoeff Ratings*

	Howard-Dolman	Verhoeff
r_{12}	0.280	0.646
r_{23}	0.338	0.758
r_{13}	0.277	0.520
Average	0.298	0.641

self reliability was found between the first and the second (r_{12}), the second and the third (r_{23}) and the first and the third (r_{13}) Verhoeff ratings than between any two of the Howard-Dolman settings. This difference in degree of self reliability between the two methods suggests that the Howard-Dolman test involves more than pure stereopsis.

The translation of both the Howard-Dolman settings and the Verhoeff ratings into terms of absolute deviations in seconds of arc [6] permits a further comparison of the results of one test with those of the other. The distribution of the adjusted readings obtained in each of the tests is given in table 4; the much greater range of

TABLE 4.—*Distribution of Averages of Three Ratings of Both the Verhoeff and the Howard-Dolman Test When These are Expressed in Terms of Absolute Deviations in Seconds of Arc*

Verhoeff Test				Howard-Dolman	
Average (Seconds of Arc)	Number of Subjects	Average Response	Number of Subjects	Average (Seconds of Arc)	Number of Subjects
3.8-3.9	16	24.0	0	0.1-0.9	42
4.0	36	25.0	0	1.0	65
5.0	35	26.0	0	2.0	37
6.0	32	27.0	0	3.0	17
7.0	16	28.0	1	4.0	10
8.0	8	29.0	0	5.0	4
9.0	10	30.0	0	6.0	2
10.0	5	31.0	0	7.0	2
11.0	4	32.0	0	8.0	3
12.0	3	33.0	0	9.0	0
13.0	4	34.0	0	10.0	1
14.0	3	35.0	0	11.0	2
15.0	0	36.0	0	12.0	1
16.0	1	37.0	2		186
17.0	1	38.0	2		
18.0	1	46.0	2		
19.0	0		186		
20.0	0				
21.0	1				
22.0	1				
23.0	2				

TABLE 5.—*Verhoeff and Howard-Dolman Ratings, Expressed in Terms of Absolute Deviations in Seconds of Arc, for Subjects Who Made the Seven Lowest Average Scores on Each Test*

Verhoeff Rating in Seconds of Arc	Subject	Howard-Dolman Rating in Seconds of Arc	Howard-Dolman Rating in Seconds of Arc	Subject	Verhoeff Rating in Seconds of Arc
28	W. T.	5.62	8	T. McG.	46.0
37	D. D.	7.26	8	J. M.	5.5
37	D. T.	5.37	8	J. P.	17.7
38	J. H.	3.04	12	R. T.	23.4
38	J. A.	3.90	11	G. J.	7.0
46	P. W.	3.16	11	M. W.	10.2
46	T. McG.	8.0	10	J. R.	11.6

6. Translation of Verhoeff readings into absolute deviations in seconds of arc was made as shown in the following computation:

Verhoeff reading, 150 cm.

2.5 mm. displacement (depth between strips at the front and those at the back of the test device)

3.2 cm. (one-half the average interpupillary distance)

$$\text{Tan } Q = \frac{150 \text{ cm.}}{3.2 \text{ cm.}} \qquad Q = 88° 46' 40.35''$$

$$\text{Tan } Q_1 = \frac{150.25 \text{ cm.}}{3.2 \text{ cm.}} \qquad Q_1 = 88° 46' 47.67''$$

$$Q_1 - Q = 7.32''$$

∴ Verhoeff reading of 150 cm. = 7.32 seconds of arc

However, in the translation of Howard-Dolman settings, the calculation was made using 10.92 mm. as equal to 2 seconds of arc. Because of the great distance from the subject's eyes, the slight variation in the angle subtended was disregarded.

ratings in the Verhoeff test suggests that this test may detect persons with poor stereopsis whose responses on the Howard-Dolman test might not be considered abnormal.

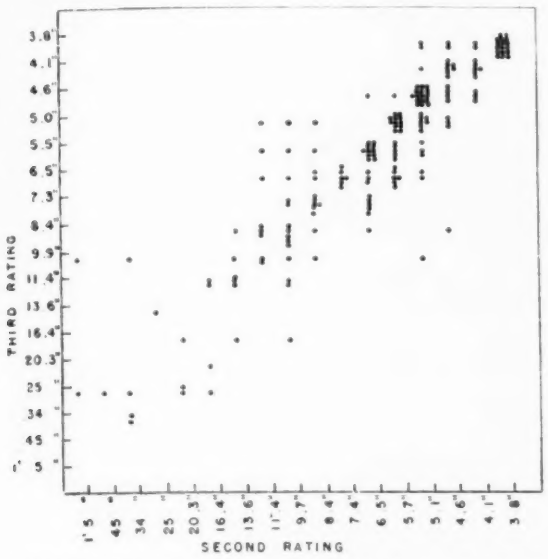

Fig. 1.—The second and the third Verhoeff rating, expressed in terms of absolute deviation in seconds of arc, plotted against each other to indicate their degree of correlation.

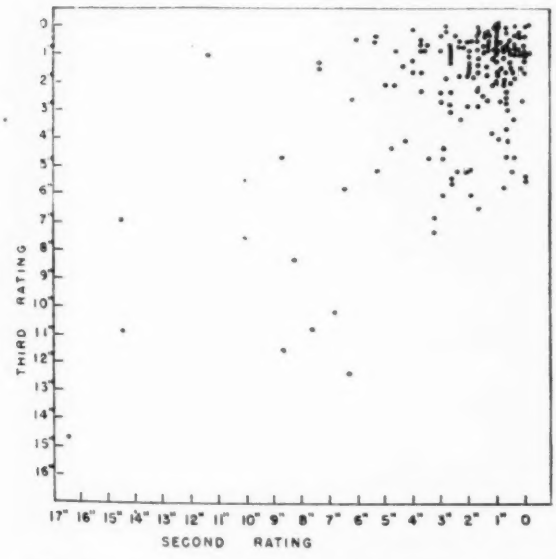

Fig. 2.—The second and the third Howard-Dolman rating, expressed in terms of absolute deviation in seconds of arc, plotted against each other to indicate their degree of correlation.

Only 1 subject (T. McG.) whose performance was among the 7 poorest on the Verhoeff test was also among the 7 poorest performers on the Howard-Dolman test (table 5). Since the nature of these two tests is such that one measures pure stereopsis while performance on the other depends on many other factors,[4] many subjects may pass the Howard-Dolman test despite a deficiency in acuity of binocular stereopsis. For the same reason, many may fail the Howard-Dolman test and yet be possessed of normal stereoscopic ability, as evidenced by their performance on the Verhoeff test.

Further evidence that a closer correlation exists between pairs of the Verhoeff readings than between pairs of the Howard-Dolman readings is graphically illustrated in figures 1 and 2. In figure 1 the second and the third response in the Verhoeff test, expressed in terms of absolute deviations in seconds of arc, are plotted against each other; and in figure 2 data from the Howard-Dolman test are similarly treated.

SUMMARY

A group of 186 subjects whose vision was previously determined to be well within normal limits in several respects was subsequently tested by both the Howard-Dolman and the Verhoeff technic for estimating stereoscopic threshold. The distributions of the averages of three responses to each of these tests are given, and the higher coefficients of self reliability between trials on the Verhoeff test are offered as evidence of the superior efficiency of this test. When the results of each test are translated into terms of absolute deviations in seconds of arc, the responses to the Verhoeff test are found to involve a much wider range than do the results of the Howard-Dolman test, a fact which indicates that the Verhoeff test is the more sensitive in detecting persons with poor stereopsis. The low correlation between the results on these two tests and the number of other factors [4] which enter into the Howard-Dolman performance support the belief that the Verhoeff test yields a superior estimate of acuity of binocular stereopsis. However, the value of the Howard-Dolman technic in the appraisal of aptitude for activities which require a number of attributes, only one among which is stereopsis, in contrast to pure stereoscopic ability, should be kept in mind.

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THE IRIDENCLEISIS OPERATION FOR GLAUCOMA

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The feeling prevails to some extent that it is not a sound surgical procedure to incarcerate iris tissue deliberately for the relief of glaucoma when on all other occasions an effort is made to prevent such an occurrence. I had occasion to review the literature on glaucoma surgery several years ago, and the reports on the iris inclusion operation were so impressive that the procedure was tried. The results have been sufficiently good to warrant continuing the use of the operation more and more during the past three years.

In this period the operation has been performed on my service on 110 eyes, and this report is based on the experience encountered in that series of consecutive operations.

An incarceration or prolapse of the iris under any circumstances may be good or bad, depending on the manner in which it occurs. After operations, especially a cataract extraction, or after perforating injuries, an incarceration may lead to a filtering cicatrix or to secondary glaucoma. If the iris is incarcerated as a wick of tissue with the surfaces of the pigment epithelium together, it is more likely to produce a filtering cicatrix. If the iris is incarcerated in such a manner that aqueous cannot drain out of the wound but dams up in a pocket of iris tissue, which tends to dilate through the effort of the aqueous to escape, then the incarceration does not lead to filtration but predisposes to glaucoma by a tendency of the iris pocket to block more and more of the filtration angle.

When an iridotasis is done, the iris should remain in a position as shown in figure 1*a*. Often, however, the sphincter muscle pulls the pupillary border of the iris partly back into the anterior chamber, as shown in figure 1*b*. This leaves a pocket of iris blocking the wound more and more because of the progressive dilation by the aqueous. This is illustrated in figure 2, which shows a microscopic section of a human eye on which an iridotasis was performed for chronic glaucoma. Incarceration of the iris in this manner is to be avoided under all conditions—at the time of an iris inclusion operation, as a complication following an intraocular operation or after a perforating injury. After a cataract extraction it is familiar how a knuckle of iris at one site in the wound may at first be so slight that it can hardly be seen but gradually becomes more and more ectatic, with perhaps some gaping of the wound. When an iris inclusion operation is performed, it is important to prevent this type of incarceration of the iris. This can best be done by employing a technic which minimizes such a possibility, and in this regard three features seem important:

1. The sphincter muscle must be cut.

2. The radial incision in the iris must reach the very periphery of the iris.

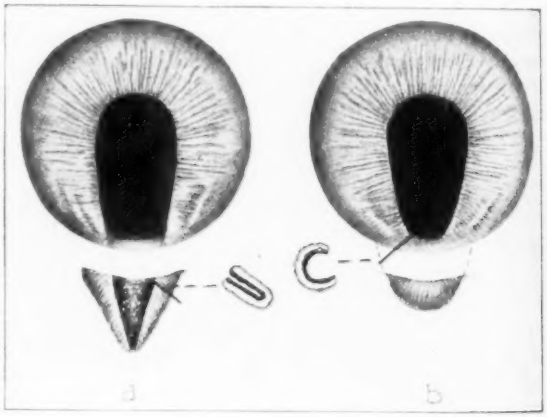

Fig. 1.—In *a* is shown the position the iris should assume after an iridotasis. The insert is a cross section of one iris pillar to show the manner in which the iris folds.

In *b* is shown the position the iris frequently assumes after an iridotasis. The sphincter muscle has retracted the pupillary margin partly back into the globe, leaving a pocket of iris. The insert is a cross section of the iris over a pillar to show the pocket formed.

This figure as well as figures 4, 5 and 9 are viewed from the surgeon's position at the head of the patient whereas figure 3 is viewed from the spectator's position.

From the Institute of Ophthalmology of Presbyterian Hospital.

Read before the New York Academy of Medicine, Section of Ophthalmology, Dec. 18, 1944.

3. There should be no root of the iris over the site of the coloboma.

The technic here described incorporates these factors.

TECHNIC

The usual injections for local anesthesia are carried out, including a retrobulbar injection of epinephrine. This is desirable not only to induce anesthesia but to lower the intraocular pressure at the time the anterior chamber is opened. It seems desirable also to wait at least five minutes after the injections are given, as absorption in glaucomatous eyes is slow. It is better to make sure of good anesthesia before the operation is started than to find out that the anesthesia is not adequate when the keratome is in the anterior chamber. A keratome section is made under a large conjunctival flap. The point of the keratome engages the conjunctiva at least 10 mm. from the limbus, and the conjunctiva is dragged down over the point of the keratome to within 2 mm. or more of the limbus, where the point then engages the sclera. The incision is completed by entering the keratome at least three-fourths the length of its blade into the anterior chamber (fig. 3). The conjunctival opening is enlarged on each side with scissors. The operator withdraws the iris by grasping it at the pupillary margin. An assistant holds the iris on the opposite side, and in unison they withdraw the iris until an iridodialysis is just noted (fig. 4 *a*). Then a radial cut is made through the iris into the iridodialysis opening (fig. 4 *b*). The assistant releases the iris, and the operator then incarcerates each pillar into the corner of the wound with a minimum amount of manipulation. The conjunctiva is closed with interrupted silk sutures. Drops are not instilled.

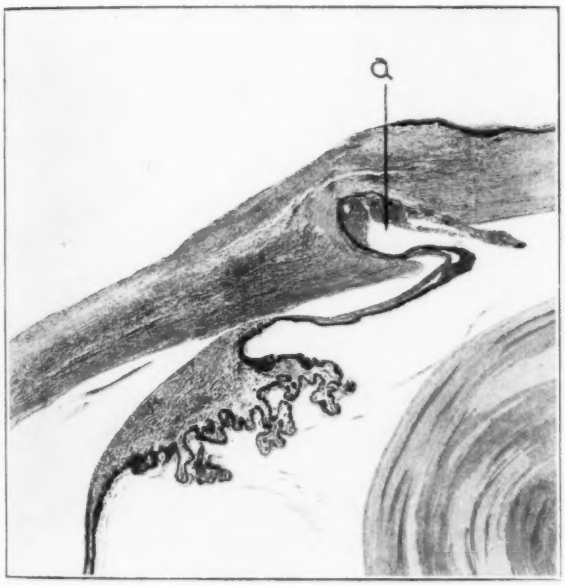

Fig. 2.—An iridotasis for chronic glaucoma. The incision is placed too far centrally. The pupillary margin of the iris has retracted into the globe, leaving the pocket of iris (*a*) dilated with aqueous.

DISCUSSION OF THE OPERATION

The most important step in the operation is a correct incision. This should be made far back so that the point of the keratome enters the anterior chamber in or close to the angle (fig. 5*a* and *a'*). This is the type of section advocated by R. G. Reese [1] for iridectomy in cases of glaucoma. If the section is placed properly in the periphery of the anterior chamber, the point of the keratome will sometimes engage the iris and push the iris ahead of it, thus producing an iridodialysis. This is desirable rather than otherwise, for it shows that the section is well placed and the iridodialysis does not have to be

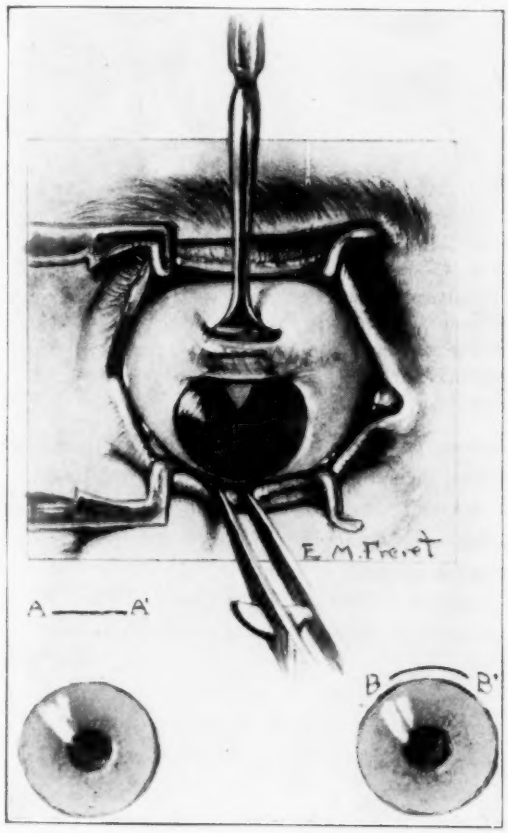

Fig. 3.—The incision with the keratome under a conjunctival flap. *A-A'* indicates the position of the incision through the conjunctiva; *B-B'*, the position of the incision through the sclera.

This figure is viewed from the spectator's position whereas figures 1, 4, 5 and 9 are viewed from the surgeon's position.

1. Reese, R. G.: A Conjunctival Flap for Glaucoma, Using a Broad Keratome, Tr. Sect. Ophth., A. M. A., 1923, p. 317.

performed later. A shelving of the cornea with the entrance of the point of the keratome in corneal tissue predisposes to failure of the operation (fig. 5*b*). Figures 2, 6 and 7 show faulty incisions in human eyes with primary glaucoma.

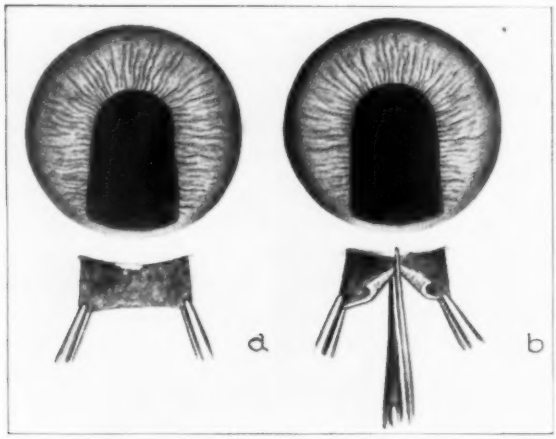

Fig. 4.—In *a*, the iris is being withdrawn until an iridodialysis is produced. The iridodialysis is shown at the center, adjacent to the wound. In *b*, the radial cut is being made into the iridodialysis opening; the stroma of the iris curls around the pigment epithelium.

Microscopic preparations show that most keratome sections for glaucoma are placed too far on the corneal side and that advantage is not taken of the wide margin of safety by making them more peripheral.

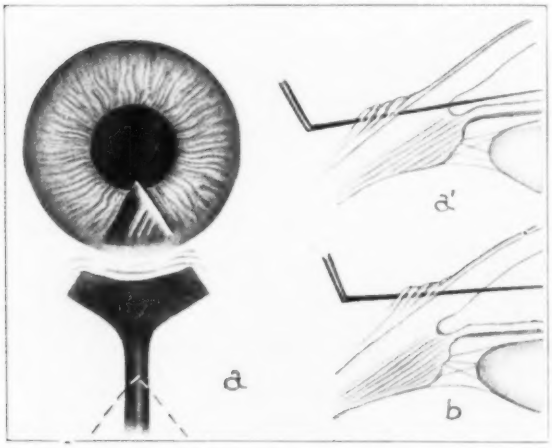

Fig. 5.—In *a*, a keratome section is made under a conjunctival flap 2 mm. or more back of the limbus. The dotted lines show the point at which the keratome engages the conjunctiva. A cross section (*a'*) shows the keratome at the proper position in the anterior chamber.

In *b*, the keratome is in the anterior chamber too far on the corneal side.

It is frequently said that the iridencleisis operation is easy, and sometimes that is a disadvantage rather than an advantage. If it were so, it would be a distinct advantage, but it is not altogether true. I should say that the operation is a short and simple procedure but not an easy one. The important step of the operation is the incision, and it is not easy to make this correctly. I have seen more ophthalmic wrecks from poorly executed keratome sections for glaucoma than I have from improperly executed trephine operations.

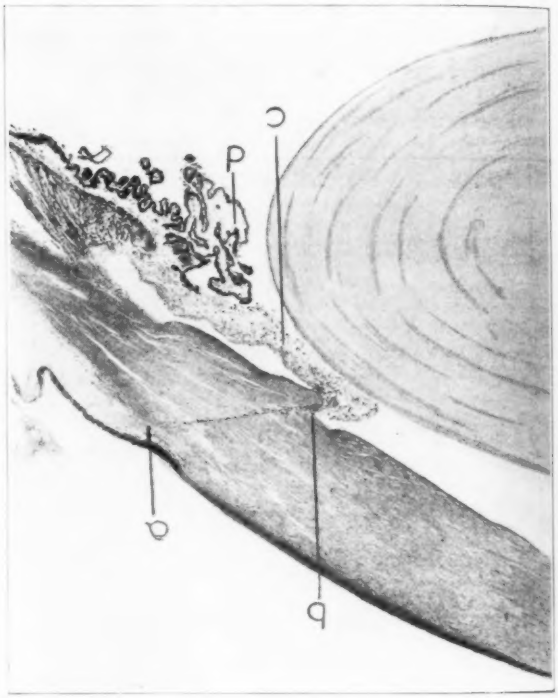

Fig. 6.—Keratome section for glaucoma extending from *a* to *b*. The section is so far on the corneal side that there is hardly sufficient iris, *c*, to reach subconjunctivally. Edematous ciliary processes are shown at *d*.

It cannot be argued that peripheral synechias prevent the execution of the section as advocated, because if the operation is carried out only in those cases in which it is indicated, as specified later, peripheral synechias will not be encountered, as they are to be expected in the later stages of glaucoma.

The conjunctival flap made with the keratome is better than the dissection of a flap because such a flap has a minimum amount of scar tissue and this is desirable for a flat, diffuse, filtering bed. The radial cut in the iris must reach the periphery of the iris to prevent the

root of the iris from blocking the angle or forming a pocket over the operative site. These aims can best be accomplished by producing an iridodialysis and having the radial cut extend into it. When the iridodialysis is produced, the iris always tears flush with the ciliary body, leaving no root, because it is here that the iris is thinnest, and also weakest, from the presence of the peripheral crypts. Figure 7 shows the manner in which the iris severs from the ciliary body when torn. It is undesirable to try to arrange the iris pillars too much, for trauma tends to

Fig. 7.—Keratome section for glaucoma extending from *a* to *b*. The section is placed too far toward the cornea. The iris was torn from its insertion into the ciliary body, at *c*. Edematous ciliary processes are shown at *d*.

stimulate the iris tissue to repair by the production of fibrous tissue. Furthermore, every precaution should be taken not to abrade the pigment epithelium. The wick of iris produced by that portion of the iris over the site of the iridodialysis seems to fall in the desired manner naturally. This is with the pigment epithelium inverted, with the curled stroma forming the outer surface (figs. 4*b* and 9 *B-B* insert). The fact that free, normal iris tissue tends to assume this position is evidenced by sections of iris tissue removed for whatever cause. In a recent case in which there was a small malignant melanoma of the iris a broad iridectomy was done. The iris was allowed to curl according to its inclination, and a section of this is shown in figure 8.

It is perhaps worth while to separate the lips of the wound by grasping the conjunctival flap at the time when the wicks of iris are placed in the angles of the incision, so that the iris tissue will be free to curl in the desired manner at the site where they pass through the wound. It is desirable to incarcerate both pillars. This seems to insure more adequate drainage and does not give hypotony.

When the operation is carried out as outlined, a section through the coloboma (fig. 9 *A-A*) should show the relations as depicted in figure 9*a*, i. e., the incision in the periphery and no iris root. A section through the iris pillar (fig. 9 *B-B*) should show the relation as depicted in figure 9*b*, i. e., the incision in the periphery and the wick of iris extending straight out the incision to the subconjunctiva. A cross section of the wick of iris in an insert opposite one of the pillars shows the pigment epithelium lining the curled stroma.

FUNCTION OF THE OPERATION

The operation is effective primarily because the wicks of iris produce a vicarious filtering sluiceway. A lesser factor contributing to the effectiveness of the operation is the iridodialysis, which frees the angle over the coloboma area. This gives the effect of a basal iridectomy. Another factor seems to be the occurrence of a taut iris below; that is, when the iris is pulled up and out of the wound above, the iris below is put on some stretch, which widens the angle. The tautness of the iris also, no doubt, tends to prevent the iris from blocking the filtration angle. This widening of the angle below, due to the tautness of the iris, is borne out by gonioscopic examination.

INDICATIONS FOR THE OPERATION

The iridencleisis operation cannot be relied on to carry too much of a drainage load. It seems to be indicated therefore in cases of glaucoma with a peak pressure of 40 to 45 or maybe 50, mm. of mercury on the Schiøtz tonometer and a basal pressure of 30 mm. of mercury or less. It is frequently not adequate in cases of glau-

coma with much higher pressure. In such instances, however, an iridencleisis operation can be combined with a sclerectomy. This was done in 3 instances and proved effective. The operation is indicated in every case of primary glaucoma in the Negro, and aside from iridectomy for true acute glaucoma, it is the only operation that is effective in persons of this race. It may be used for secondary glaucoma in eyes with inactive uveitis. It has been surprising how well the operation is tolerated in eyes in which the uvea was the seat of an inflammation. In 2 instances the inflammation seemed not entirely inactive, but still the operation was effective and well tolerated. It seems as effective as any other operation in cases of hydrophthalmos and buphthalmos.

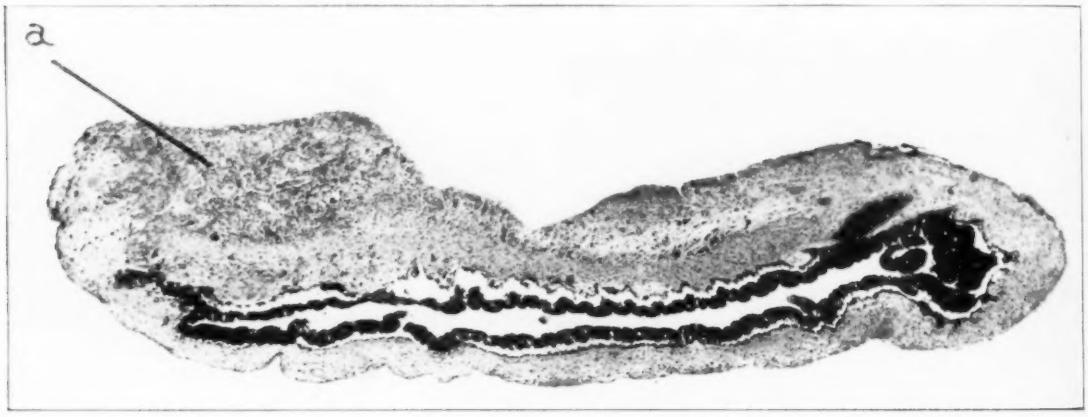

Fig. 8.—Normal iris curling to form a wick, the central lumen of which is lined with pigment epithelium. (The specimen is a portion of iris removed because of a malignant melanoma. The periphery of the tumor is shown at *a*.)

CONTRAINDICATIONS TO THE OPERATION

In cases in which one or more operative procedures have failed, the use of the iridencleisis operation has not proved satisfactory. This may be due to the fact that in most instances the drainage load necessary for the operation to carry is too great. It also is not satisfactory in cases of long-standing glaucoma in which not only is the pressure high but secondary changes in the iris, such as vascularization and atrophy, have occurred. In glaucomatous eyes the iris stroma is replaced by connective tissue as the disease advances, and such irises when incarcerated probably produce fibrous tissue, which tends to close the filtering area. A relatively normal iris has little or no tendency to the formation of repair tissue. I have never used the operation in cases of acute glaucoma because for them iridectomy is adequate. In cases of acute exacerbation of chronic glaucoma, the operation could be used if none of the contraindications is present. It is not indicated in cases of glaucoma in aphakic eyes.

RESULTS

Statistics on glaucoma are difficult to evaluate and compare because so much depends on the stage of the disease at which the operation is done, on the duration of the follow-up period, on the type of glaucoma and on whether or not the operation is primary or secondary to other operations.

As the indications for the operation became more apparent, it has been restricted more and more to cases of the type indicated in this paper.

The statistics presented here are based on 61 operations which I performed on private patients, as the records of these patients lend themselves better to analysis.

The disease was considered arrested if the postoperative intraocular pressure was never higher than 27 mm. of mercury with the Schiøtz tonometer one month after the operation. The results were reckoned after the lapse of this postoperative period because in a few instances it was noted that the intraocular pressure fluctuated somewhat above normal for a few weeks after operation and then leveled off to normal and remained there. In 1 instance the postoperative pressure was found to be above normal several times for a postoperative period of two months and then became normal.

With these criteria, the disease was deemed arrested in 52 of the 61 eyes, or in 85 per cent.

It is true that some of the patients have not been followed for a sufficiently long postoperative period.

An analysis of the 9 cases of failure shows that in 7 of them the operation was contraindicated according to the stipulations in this paper. These 9 cases may be summarized as follows:

Case 1.—Secondary glaucoma with retinal detachment from retinitis proliferans. The intraocular pressure was 70 mm. of mercury. The eye was blind and painful. Permission for enucleation was refused.

Case 2.—Secondary glaucoma from an iridocyclitis which was somewhat active.

Case 3.—Primary glaucoma, for which four trephine operations had been done before the iris inclusion operation.

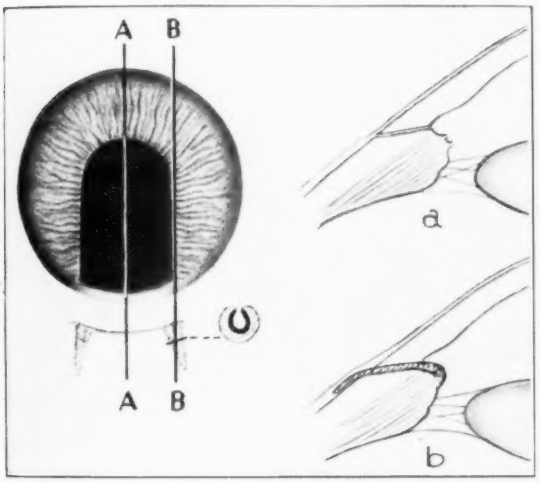

Fig. 9.—A section through the coloboma at *A-A* would appear as shown in *a*. A section through the pillar at *B-B* would appear as shown in *b*. The insert shows a cross section of the wick of iris.

Case 4.—Primary glaucoma in a Negro. One trephine operation had been done before the iris inclusion operation.

Case 5.—Same patient as that in case 4. A second iridencleisis was done.

Case 6.—Primary glaucoma, for which a trephine operation failed before the iris inclusion operation was done.

Case 7.—Primary glaucoma with an intraocular pressure of 70 mm. of mercury. The iris was semidilated and immobile from atrophy.

Case 8.—Primary glaucoma with no contraindication to the operation. The failure of the operation was attributed to a poorly placed incision.

Case 9.—Primary glaucoma with no contraindication to the operation. The failure of the operation was attributed to a poorly placed incision.

If the intraocular pressure was as high as 27 mm. of mercury at any time during the follow-up period, a miotic was prescribed as a precautionary measure, to be used twice daily. This was necessary in 29 per cent of the cases of arrested glaucoma. Of the cases in which the operation was successful, the difference between the mean of all of the preoperative pressures and the mean of all of the postoperative pressures was 18.7 mm. of mercury, i. e., the operation effected a mean reduction of 18.7 mm. of mercury in the intraocular pressure. It has been stated that the operation is indicated in cases with a peak pressure of 45 and perhaps 50 mm. of mercury. Subtracting the mean reduction of 18.7 mm. from 50 mm. leaves 31.3 mm. Selecting 45 to 50 mm. of mercury as the peak pressure is, therefore, on the conservative side, because the peak pressure is appreciably higher than the mean pressure.

ADVANTAGES OF THE OPERATION

I believe that the best way to arrest glaucoma is to operate at the earliest possible time [2] after it is clear that an operation is indicated. This usually means that the operation is done when the patient still has normal vision and normal, or almost normal, fields. The large majority of patients with glaucoma are practically symptomless. Therefore it is usually necessary to advise an operation on a patient with normal vision, normal fields and no symptoms. The patient is offered no tangible result, as is the patient with cataract or squint. He is offered only the hope that the vision can be saved for the future. When I advise an operation in such a situation, I like to feel not only that there is an excellent chance of arresting the disease but that there is a negligible chance of any complications being created. This is definitely my feeling in regard to the iridencleisis operation, but not so in regard to the trephine operation, which is too frequently followed by quiet iritis, slow restoration of the anterior chamber, detachment of the choroid, hypotony, a redundant filtration bleb, progressive changes in the lens or late infection.

There are really no serious complications following an iridencleisis operation. Sometimes mild iritis manifests itself as a slight proliferation of pigment around the pupillary margin and a

2. Reese, A. B.: The Value of Early Operation in Chronic Primary Glaucoma, J. A. M. A. **113**:1204 (Sept. 23) 1939.

slight tendency to posterior synechias. This feature, however, has never proved serious. Sometimes there is a hyphemia, but this absorbs and has never caused any untoward consequences. The hyphemia may be the cause of a somewhat elevated intraocular pressure for a time after the operation.

Eyes with primary glaucoma are more predisposed to the development of cataract than nonglaucomatous eyes. One third or more of the eyes with primary glaucoma have incipient cataractous changes. Operations for glaucoma in general, and especially the trephine operation, tend to hasten the development of these incipient cataractous changes. An analysis of the cases in which the iridencleisis operation has been done seems to indicate that this procedure predisposes less to the progression of incipient cataractous changes than any other operation for glaucoma except the straight iridectomy. In no case has hypotony, detachment of the choroid, slow restoration of the anterior chamber, a redundant filtration bleb or late infection been noted.

DISADVANTAGES OF THE OPERATION

The possibility of sympathetic inflammation is frequently mentioned in connection with this operation. This complication has not appeared in the present series of cases, nor has it occurred in a series of over 200 cases at the Illinois Eye and Ear Infirmary.[3] Every intraocular operation has a very high incidence of incarcerated uveal tissue of some degree and a very low incidence of sympathetic inflammation. From my experience alone I have no reason to feel that the incidence is any higher in the one intraocular operation than in the others. I am inclined to believe, however, that in the long run the incidence of sympathetic inflammation must be somewhat higher after the iris inclusion operation than after other intraocular operations but that it is not sufficient to be a serious contraindication to the operation.

Sometimes the question of a pulled-up, or hammock, pupil is mentioned. There is a tendency for the pupil to be higher, but at no time has this feature been sufficiently pronounced to be of consequence.

A cataract extraction is perhaps made somewhat more difficult by this type of operation.

Also, if a trephine operation is done on an eye which has been subjected to an iris inclusion operation, the iris tends not to prolapse after the trephining if the site of the operation is over one of the iris pillars. This is due to the fact that the tautness of the pillar prevents the iris from prolapsing to any extent, if at all.

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DISCUSSION

DR. ARNOLD KNAPP, New York: Several things in Dr. Reese's demonstration interested me particularly. I had long had the idea that one of the advantages of the iridencleisis operation was that it was an easy procedure, as the section was made at the limbus, but Dr. Reese stresses the making of the incision 2 mm. back of the limbus, a method which in the chronic glaucomatous eye is often very difficult. Furthermore, Dr. Reese produces an iridodialysis and believes that he is tearing the iris at its root. With chronic glaucoma, however, the angle of the anterior chamber is obliterated, and one cannot get at the root of the iris. Dr. Reese has not made clear why he has abandoned the operations for chronic glaucoma which have given such good results — the Lagrange and the modified trephine operation. I have always felt that iridencleisis should be done as a last resort, and then combined with sclerectomy.

DR. KAUFMAN SCHLIVEK, New York: When the iridencleisis operation was introduced in Vienna, several years ago, I saw many cases and was much discouraged with the results. A few years ago I saw the operation performed in another hospital in a case in which sympathetic ophthalmia developed. My experience with this operation has been slight. In the few cases in which my associates and I have performed it the results have not been satisfactory, but in our institution we always follow the rule: acute glaucoma: iridectomy; secondary glaucoma: Lagrange operation; chronic simple glaucoma: trephination or, if the iris is atrophic, trephination with complete iridectomy.

DR. DAVID H. WEBSTER, New York: I have limited this operation to cases of infantile glaucoma. It has been the only procedure in which I have succeeded in controlling the tension and retaining vision. In cases of infantile glaucoma the iris is elastic except those in which the disease is accompanied with other degenerative changes and no operation seems to succeed, but in the average case of infantile glaucoma, in which the cornea

3. Kronfeld, P.: Personal communication to the author.

measures from 15 to 16 mm., even though the cornea is milky white, the iris can easily be grasped, pulled into the wound, separated at the root and incised and the divided pillars left under the conjunctiva.

Dr. Robert K. Lambert, New York: During the past year my colleagues and I have employed iridencleisis much more frequently than was formerly done on the eye service at Mount Sinai Hospital, and we have usually had good results.

Dr. John McLean, New York: I have been seeing and doing this operation a little longer than Dr. Reese, for about ten years, during which time I have seen, though they were not mine, 3 cases of sympathetic ophthalmia following iridencleisis. I have also seen cases of sympathetic ophthalmia following iridectomy, trephining and cataract extraction. The incidence following iridencleisis has been sufficiently low not to influence me to discontinue the use of this operation, but I am sure that if Dr. Reese continues to do this operation he will encounter the complication sooner or later.

As to the ease or difficulty of the operation, with the keratome incision (which is only one of several ways in which it may be done) there will not be much difficulty if the cases are limited to those indicated by Dr. Reese as suitable, among others, those of early glaucoma in which the tension has not become too high, for in most such cases there will not be very shallow chambers or extensive anterior peripheral synechias and there will be room to make a fairly easy keratome incision, about 1.5 to 2 mm. back of the limbus.

I should like to make one other suggestion, namely, that another good reason for doing this operation in suitable cases is that for the occasional operator it is really much easier than trephining, or even the various modifications of the Lagrange operation.

Dr. Henry Minsky, New York: I should like the privilege of making a few remarks, especially since Dr. Reese pointed out many years ago, as he did tonight, that the selection of the site of the keratome incision is one of the important steps in the operation. Also, Dr. Knapp has raised the question of the difficulty of the keratome incision, as has Dr. McLean, in cases of the late stages of glaucoma. For years I have taught a simple method of selecting the site of the keratome incision which makes the operation easy for the novice, as well as for an experienced operator. If the contralateral point on the limbus is illuminated by focal illumination, as is done in examination with the slit lamp, a brilliant crescent appears at about 12 o'clock at the extreme periphery. If the point of the keratome is entered in the sclera at the very edge of the illuminated crescent and the keratome sent home in the plane of the iris, the surgeon will inevitably hit the angle of the anterior chamber just anterior to the ciliary processes and perform the peripheral incision at the limbus, so as to get the root of the iris without fail. If that is done, the necessity of tearing the root of the iris may perhaps be avoided, for when the incision is made in the manner suggested the iris is easily grasped, pulled into the wound and put into position.

The mere specification of 1.5 or 2 mm. from the limbus is not sufficient, because the measure is an arbitrary one and the position of the limbus varies in each eye, especially in the glaucomatous eye. One can easily find the precise angle of the anterior chamber and make the iridencleisis more successful by the method of the incision suggested.

Dr. Algernon B. Reese, New York: Dr. Knapp said that one of the virtues of the operation was that it is easy to perform. The procedure is a simple one, but I do not believe it is particularly easy to make a correct incision far in the periphery of the anterior chamber — and this is the most important feature of the operation.

Dr. Knapp also stated that in the eye with chronic glaucoma the angle is obliterated and therefore one cannot produce an iridodialysis as described. If there are peripheral synechias, the operation is not indicated, for synechias do not occur except in advanced glaucoma. I think synechias are likely to be the exception in the eyes which are operated on for glaucoma in general, and if they are present the glaucoma is in an advanced stage. In cases of this type some other operation which will carry a greater drainage load is indicated.

I have not given up the trephine, cyclodialysis and Lagrange operations but use them only in indicated cases.

Dr. Schlivek brings up the question of sympathetic inflammation. This presentation is based on my personal experience with the operation. In this series of 110 operations we have had no case of sympathetic inflammation. The incidence of this complication may be higher in this operation than it is in other intraocular procedures, but my experience does not bear this out.

Dr. Schlivek states that he has not done this operation on many eyes. I think this is in accord with the experience of most of us in this section. We have been loath to adopt this operation, and in some measure it is heresy to expound the virtues of the operation.

Dr. Webster mentioned that the operation is useful in cases of hydrophthalmos. Dr. Sanford Gifford also expressed this view in an article several years ago. I have performed the operation in 2 such cases; in both it worked well. There were, however, rather large redundant blebs in both instances.

Dr. McLean also brings up the question of sympathetic inflammation. I agree with him that sooner or later I shall encounter sympathetic ophthalmia as a consequence of the operation. In the meantime, however, I hope to have arrested more cases of glaucoma.

Dr. McLean also states that the operation is an easy one. If so, this is a virtue and not a fault. I contend it is a simple procedure but not an easy one to execute correctly. I believe more eyes are ruined by a poorly executed keratome section for glaucoma than by the trephine operation.

Dr. Lambert mentions some good results he has had with the operation.

Dr. Minsky brings out a good point regarding the placing of the incision.

EFFECT OF TALC IN OCULAR SURGERY

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Since rubber gloves have become popular in the operating rooms of many ophthalmic institutions, one is prompted to study the effect on the ocular tissues of the talc powder on these gloves. There has been a great deal of investigation along these lines, starting in 1933, when Antopol [1] reported the formation of tubercles by spores of Lycopodium. At that time the spores of Lycopodium were used on surgical gloves, as talc is used today. However, even as far back as 1912, Lambert [2] reported a foreign body giant cell reaction to spores of Lycopodium. He used the substance as a nonspecific foreign body and stated that he could just as well have used cotton fibers, or even glass.

In later years, when talc had largely replaced spores of Lycopodium, similar investigations were conducted by various workers with the express purpose of determining the effect that accidental introduction of talc from surgical gloves produces on tissues. The main constituent of talc is magnesium trisilicate, which composes about 60 per cent of the substance. Correspondence with various manufacturers revealed fairly uniform qualitative and quantitative analyses of the talc powders. The talc used in the present study was obtained from Chas. B. Crystal Co., Inc., who furnished the following quantitative analysis:

	Per Cent
Silica (magnesium trisilicate)	60.04
Aluminum oxide	1.80
Iron oxide	0.16
Calcium oxide	0.38
Magnesium oxide	30.98
Loss on ignition	6.22

In general, the various investigators in the field have reported fairly uniform results. The crystals of magnesium trisilicate were found in large granulomatous masses which were produced by the experimental introduction of talc in animal and human tissues. Such observations have been reported by Ramsey and Douglass,[3] de Savitsch,[4] Byron and Welch,[5] German [6] and several others.

The granulomas produced by talc were found to cause the clinical picture of tumor, the mass being even large enough to produce intestinal obstruction in some cases. In other cases visceral adhesions were formed. In view of such observations, it seemed important to determine whether similar reactions occur in the eye, as no such studies have been reported. Tumors of any size produce more serious sequelae in a compact, enclosed organ such as the human eye, in which all physiologic functions are dependent on fine anatomic structures.

One series of studies consisted of the withdrawal of varying amounts of aqueous from rabbit eyes and the replacement with similar amounts of suspension of talc of varying concentration in isotonic solution of sodium chloride. The second series consisted of iridectomies with irrigation of the anterior chamber with suspension of talc in saline solution, in two concentrations. In the third series, varying amounts of a suspension of talc were injected between the extraocular muscles and the sclera. By using these three methods of introduction of talc my associates and I planned to simulate the accidental introduction of talc into ocular tissues during surgical procedures.

TECNIC

In order to avoid extraneous factors, the procedures were made as simple as possible and were carried out with local anesthesia and with aseptic precautions. The rabbit was placed in a small wooden operating box, and the assistant immobilized the head with one hand

From the Ophthalmological Service of Montefiore Hospital for Chronic Diseases.

1. Antopol, W.: Lycopodium Granuloma, Arch. Path. **16**:326-331 (Sept.) 1933.

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3. Ramsey, T. L., and Douglass, F. M.: Granulomatous Inflammation Produced by Foreign Body Irritants, J. Internat. Coll. Surgeons **3**:3-10 (Feb.) 1940.

4. de Savitsch, E.: Granuloma from Penetration of Talcum Powder, M. Ann. District of Columbia **9**:169-170 (May) 1940.

5. Byron, F. X., and Welch, C. S.: Complications from Use of Glove Powder, Surgery **10**:766-769 (Nov.) 1941.

6. German, W. M.: Dusting Powder Granulomas Following Surgery, Surg., Gynec. & Obst. **76**:501-507 (April) 1943.

and with the other inserted a thin metal tongue depressor outside the lower lid, between the globe and the orbital floor. With exertion of firm, but gentle, pressure toward the apex of the orbit, the globe was proptosed. Several instillations of 0.5 per cent tetracaine hydrochloride were made before and after proptosing the eye. If the tongue depressor was kept in place, the globe not only remained proptosed but was immobilized. This approach made the eyes accessible to our procedures. No general anesthetic was used. In performing the iridectomies with local anesthesia, we were concerned about the pressure exerted in proptosing the globe with the assistant's tongue depressor. However, once the globe was proptosed and the pressure with the tongue depressor somewhat relaxed, the globe rotated into its "anatomic" position and remained proptosed, while the tongue depressor was kept loosely inserted between the globe and the lower orbital wall, with practically no pressure exerted on the eyeball.

PROCEDURES AND RESULTS

First Series: Introduction of Talc into Anterior Chamber.—Three rabbits were used in this series of studies. Varying amounts of aqueous were withdrawn from the anterior chamber into a syringe and replaced with similar amounts of suspensions of talc, of varying concentrations, in isotonic solution of sodium chloride. In the control eye, saline solution was used instead of the suspension of talc. As the table indicates, all the eyes in which suspension of talc was injected showed reactions of varying degrees. At first, the talc settled to the lower angle of the anterior chambers in a crescent-shaped distribution. This mass was eventually replaced with a granuloma in 3 eyes. Two eyes showed only a number of foreign body giant cells under the endothelium of the iris. That portion of the cornea which was in contact with the talc exhibited some vascularization. The control eye, 5R, did not show any of these inflammatory reactions.

Second Series: Iridectomy with Irrigation of Anterior Chamber with Suspension of Talc.—In this series, 4 rabbits were used, the iridectomy being performed on one eye of each animal. In 3 of the eyes the anterior chamber was irrigated with a suspension of talc, and in the control eye saline solution was used. It is interesting to note that yellowish exudate was formed at the site of the coloboma. That is, the foreign body reaction was more pronounced in those tissues which had undergone surgical trauma than in the lower angle of the anterior chamber, to which the talc gravitated in the first series and in which no intraocular operation was performed. This observation coincides with the experience of German.[6] This author found that foreign body granulomas due to talc were more apt to form on tissues that had undergone surgical trauma than on smooth serous surfaces. In the control rabbit there were no exudates of any kind. In all these rabbits the use of atropine or any other drug was avoided so as not to influence the postoperative course more than was necessary.

Third Series: Submuscular Injection of Talc.—In this series, 3 eyes were used. Actual operation on the muscle was avoided so that the reaction should depend as much as possible on the talc alone. Varying amounts of suspension of talc were injected under the superior rectus muscle in each eye. This was done through a small nick in the conjunctiva and Tenon's capsule, at a little distance from the muscle. A needle of the type used to irrigate the lacrimal sac was introduced along the smooth scleral surface to a position beneath the belly of the muscle, which was easily visualized through the thin conjunctiva. In all cases granulomatous masses were produced beneath the muscles.

ENUCLEATIONS

Enucleations were carried out with intravenous injection of veterinary pentobarbital sodium one month after the initial procedures. Most of the animals required 5 cc. of veterinary

Data on Experimental Introduction of Talc in Rabbit Eyes

Rabbit and Eye No.	Procedure: Aqueous Withdrawn, Cc.	Procedure: Amount of Talc Suspension Injected Into Anterior Chamber	Clinical Reaction in 14 Days *	Histologic Reaction †
Series 1: Talc in Anterior Chamber				
4R	0.05	0.05 cc. of 0.03%	+++	+++
4L	0.10	0.10 cc. of 3.0%	++++	++++
5L	0.10	0.10 cc. of 0.5%	+	++
7R	0.20	0.10 cc. of 3.0%	++++	++++
7L	0.10	0.10 cc. of 0.5%	+	+
5R	0.10 (control)	0.10 cc. of 0.5%	—	—
Series 2: Iridectomy and Irrigation with Talc				
1R	Irrigation of anterior chamber with 0.5% talc suspension.....		+++	+++
3R	Irrigation of anterior chamber with 1.0% talc suspension.....		++	++
9R	Irrigation of anterior chamber with isotonic solution of sodium chloride (control)		—	—
Series 3: Talc Injected Submuscularly				
10R	0.3 cc.	of 10 % talc suspension	+++	++++
11R	0.2 cc.	injected between muscle	++	+++
11L	0.3 cc.	and sclera	+++	+++

* In this column, — indicates no inflammatory reaction; +, a slight inflammatory reaction (uveal or submuscular); ++, moderate inflammatory reaction (uveal or submuscular); +++, moderate inflammatory reaction, with mass in the anterior chamber or under the muscle, and ++++, marked inflammatory reaction, with large mass in anterior chamber or under the muscle.

† Here, — indicates no reaction; +, foreign body giant cells under endothelium of the iris; ++, foreign body giant cells and fibrosis; +++, moderate foreign body granuloma formation, and ++++, large foreign body granuloma formation.

pentobarbital sodium (0.065 Gm. per cubic centimeter) for the enucleation. The eyes were fixed in Bouin's solution and, after the usual dehydration with alcohol and xylene, were embedded in paraffin and stained with hematoxylin and eosin.

COMMENT

In studying the table, one can readily see that talc did produce foreign body granulomas in the majority of the eyes. Three of the 5 eyes (7 R, 4 R, 4 L) which had talc inserted into the anterior chamber showed formation of granulomatous masses (fig. 1). In the other 2 eyes some foreign body giant cells were noted under the endothelium of the iris, together with swelling of the ciliary epithelium. In the second series, the 2 eyes which were irrigated with a suspension of talc after iridectomy showed a foreign body giant cell reaction, with a granulomatous mass in 1 of them (1 R). The control eye (9 R) did not show any foreign body giant cells. In the third series, all 3 eyes which had talc inserted under the muscle showed a foreign body granuloma at the site of injection (fig. 2).

Fig. 1 (eye 7R).—Foreign body granuloma in the angle of the anterior chamber produced by injection of talc into the chamber. *A* indicates the cornea; *B*, a foreign body granuloma in the angle of the anterior chamber, with fibrotic adhesions to Descemet's membrane, anteriorly, and to the iris, posteriorly; *C*, the iris, and *D*, the cilioscleral sinus, first described by Troncoso (ARCH. OPHTH. **31**:481-502 [June] 1944).

Fig. 2 (eye 10R).—Foreign body granuloma on the outer surface of the sclera. *A* indicates a granulomatous mass under the subconjunctival tissue; *B*, episcleral tissue; *C*, the sclera, and *D*, the ciliary body.

Our observations show that the extent of the reaction, as observed clinically and histologically, does not necessarily correspond to the quantity of talc introduced into the eye. For instance, as seen in the table, eye 4 R in which less talc was inserted, showed a far greater reaction than did eye 5 L, which received a greater amount of talc. Furthermore, when the aqueous was completely removed prior to the introduction of talc, the reaction was most severe. I ascribe

this to the greater protein content of the secondary aqueous. As a matter of fact, in preliminary studies, in which varying amounts of aqueous were withdrawn from rabbit eyes, in general the more aqueous was withdrawn the greater tendency there was to formation of fibrin coagulum. Therefore, when the anterior chamber was completely empty, the protein content of the secondary aqueous was greatest and a more severe reaction followed.

Microscopic study of the granulomatous masses revealed a similar cytologic picture in most of the eyes. Essentially, the reaction was that of an inflammatory mass, consisting of polymorphonuclear leukocytes, talc crystals (fig. 4), foreign body giant cells, some small round cells and fibrous tissue. The foreign body giant cells (fig. 3) contained large talc crystals or debris from talc in various states of disintegration. Varying amounts of fibrin were found along the outer layers of the granulomas, binding the mass to the adjacent tissues. Thus, in

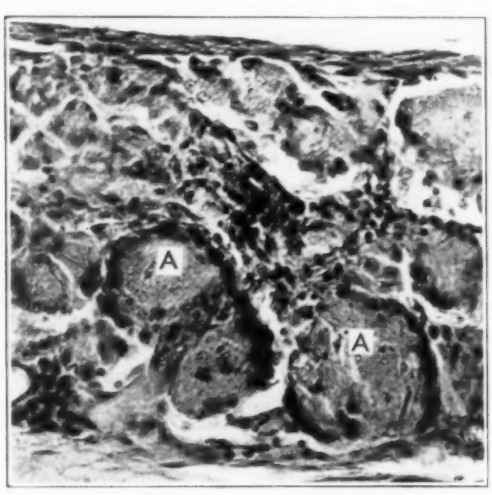

Fig. 3 (eye 10R).—Multinucleated foreign body giant cells from a granuloma under muscle. *A* indicates multinucleated giant cells, with disintegrating talc within. × 200.

the angle of the anterior chamber, one found fibrin binding the mass to corneal endothelium, on one side, and to the anterior surface of the iris, on the other, with destruction of iris tissue by scar formation (fig. 1). The swelling of the ciliary epithelium was fairly uniform in all the eyes in which talc was introduced intraocularly. In the eyes with least reaction, eye 5 L for example, only some foreign body giant cells were found beneath the anterior endothelium of the iris, without any definite mass formation.

In eye 7 R, in which the clinical reaction was greatest, the granuloma was largest and protruded into the anterior chamber about one-third the distance across and extended, to a lesser depth, around the entire circumference of the angle (fig. 1).

In the series of iridectomies, the foreign body tissue reaction bridged the coloboma in eye 1 R. In eye 3 R fibrous tissue was laid down at the site of iridectomy, although there was no complete bridging of the coloboma. In eye 9 R, the control, there was no evidence of foreign body giant cells or of fibrous tissue formation of any

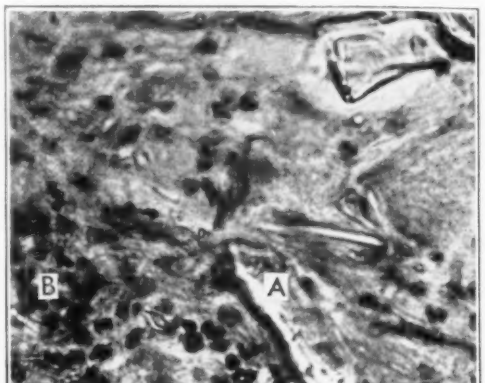

Fig. 4 (eye 7R).—Talc crystals in a foreign body granuloma in the anterior chamber. *A* indicates talc crystals and *B*, polymorphonuclear leukocytes. × 200.

kind. This is in accordance with the observations of Daniel,[7] who showed that there was no permanent regrowth of iris or fibrous tissue following iridectomy in the rabbit's eye.

In the studies on submuscular injection, gross dissection revealed definite adhesion of muscle to sclera at the site of the granulomatous masses. Microscopically, these masses showed the same histologic structure as the granulomas in the anterior chamber.

In evaluating the practical significance of these reactions, one might ask how much talc can actually get into the tissues with the usual operative procedures. The answer is that as long as there is a possibility that any can get in, the danger should be avoided.

In surgical procedures on muscles, especially, the operative field is widely exposed in contrast to intraocular operations, such as cataract extraction, which in most instances are done through a small wound.

In surgical procedures for glaucoma, such as trephination, sclerectomy and other types of

7. Daniel, R. K.: Healing of the Iris in Rabbits Following Experimental Iridectomy, Arch. Ophth. **31**: 292-298 (April) 1944.

filtering operations, it is easily conceivable that some talc may fall over the site of sclerectomy, or at the areas of inclusion of the iris. Certainly, it would be undesirable to have a foreign body granuloma formed at such a site. A granuloma here may easily occlude the tiny openings on whose patency depends the success or failure of the operation.

In muscle surgery, one is prompted to investigate the granulomatous masses often seen for many weeks at the site of operation. Perhaps one should have more proof before ascribing these masses to the type of suture material used or to the amount of resection, because it is conceivable that some of them might be foreign body granulomas produced by talc. It would be worth while to investigate such masses histologically.

To remove powder from gloves, it is best to have the assistant play the stream of saline solution from an irrigator over the operator's gloved hands before he starts working. The use of the irrigator for this purpose avoids the need for frequent change of the solution of mercury bichloride or cresol in the basin and assures one of a constant stream of clean saline solution, instead of one contaminated by the preceding surgeon's talc washings.

In the recent literature, there has been a good deal of discussion on various substitutes for talc. The two substances most favorably received thus far are potassium bitartarate [8] and hydrolyzed starch.[9] The potassium bitartarate is supposed to be rather easily absorbed by body fluids, instead of causing foreign body granuloma. Neither substance was easily available during the period of the war. Certainly, one of these, or, perhaps another substance, will be found to be a satisfactory substitute for talc. Before the new powder is adopted for use in surgical procedures, however, it would be well to carry out studies similar to those described with talc.

SUMMARY

Talc has been demonstrated to produce granulomas in the eyes of rabbits. When rubber gloves are used, it is advisable to remove all the talc before operating.

1840 Grand Concourse.

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9. Seelig, M. G.: Dusting Powder for Surgical Gloves, J. A. M. A. **125**:1208 (Aug. 28) 1944.

SCLERAL FLAP INCISION WITH SCLERAL SUTURES FOR THE CATARACT OPERATION

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Ophthalmologists have given much time and thought to the perfection of the cataract operation. A considerable literature on the technic of cataract surgery has accumulated in recent years, much of it concerning accurate and firm closure of the incision.

It has been generally accepted that firm closure of the cataract wound leads to fewer postoperative complications, such as prolapse of the iris and vitreous, fewer cases of delayed closure of the anterior chamber and a considerable decrease in the incidence of postoperative hyphemia. The experimental studies of Hilding [1] and the statistical studies of Leech and Sugar [2] and McLean [3] all bear this out. Firm closure of the incision allows the patient much more freedom of movement and permits the aged patient to be out of bed forty-eight hours, or even as early as twenty-four hours, after the operation. It is evident that this is quite an achievement when one considers the weakening effect of a prolonged stay in bed. Furthermore, postoperative astigmatism is found to be at least a full diopter less than when no sutures are employed.

Because of ease of application, the method of conjunctival suture introduced by Williams,[4] in 1869, finds many adherents. Especially is this true of ophthalmic surgeons who feel that the presence of a preplaced suture, particularly the corneoscleral suture, might interfere with the making of a good incision. However, the conjunctival suture, while it is of definite value, offers the patient and the surgeon little more in the way of security than does no suture at all.

Recently the corneoscleral suture has become more commonly employed.[5] This suture, first introduced by Kalt [6] in 1894, with its various modifications since that time, is designed to be placed before the cataract incision is made. As its name implies, it consists of two parts, the corneal and the scleral, between which the cataract knife or keratome must pass in making the incision.

There are several drawbacks to use of the corneoscleral suture. If the corneal and the scleral part are placed close together, with an interval of 0.5 mm., as recommended by Kalt, it definitely interferes with the making of the incision. I have observed that the corneal and the scleral part of this suture are more likely to be placed 2 to 3 mm. apart, in which case the lips of the wound are less firmly and less accurately brought together. Tying the suture too tight in such a case invariably causes inversion and buckling of the lips of the wound. Further, the wound is not well covered with conjunctiva unless a Kuhnt conjunctival flap is first made and sutured over it.

McLean,[3] in 1940, described a corneoscleral suture which he places through a groove made at the limbus under a small conjunctival flap. This suture overcomes the aforementioned drawbacks, except that it requires the utmost accuracy to pass the Graefe knife between the lips of the groove and, at the same time, to avoid cutting the suture. Even if one uses the keratome and scissors for making the incision, the suture may easily be severed.

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1. Hilding, A.: Efficiency of Various Wound Closures in Prevention of Prolapse of the Iris After Cataract Operations: Experimental Study, Arch. Ophth. **22**:177 (Aug.) 1939.

2. Leech, V. M., and Sugar, H. S.: Reduction of Postoperative Complications in Cataract Operations with Corneoscleral Sutures, Arch. Ophth. **21**:966 (June) 1939.

3. McLean, J. M.: A New Corneoscleral Suture, Arch. Ophth. **23**:554 (March) 1940.

4. Williams, H. W.: Cataract Extraction Operations, Arch. Ophth. **1**:98, 1869.

5. Ellett, E. C.: Use of Suture in Extraction of Cataract, Arch. Ophth. **17**:523 (March) 1937. Stallard, H. B.: A Corneoscleral Suture in Cataract: Its Technique and Advantages, Brit. J. Ophth. **22**:269, 1938. Verhoeff, F. H.: A Corneoscleral Conjunctival Suture in Operations for Cataract, Tr. Am. Ophth. Soc. **25**:48, 1927. Walker, C. B.: Exactly Appositional Sutures in the Cataract Operations, ibid. **27**:51, 1929.

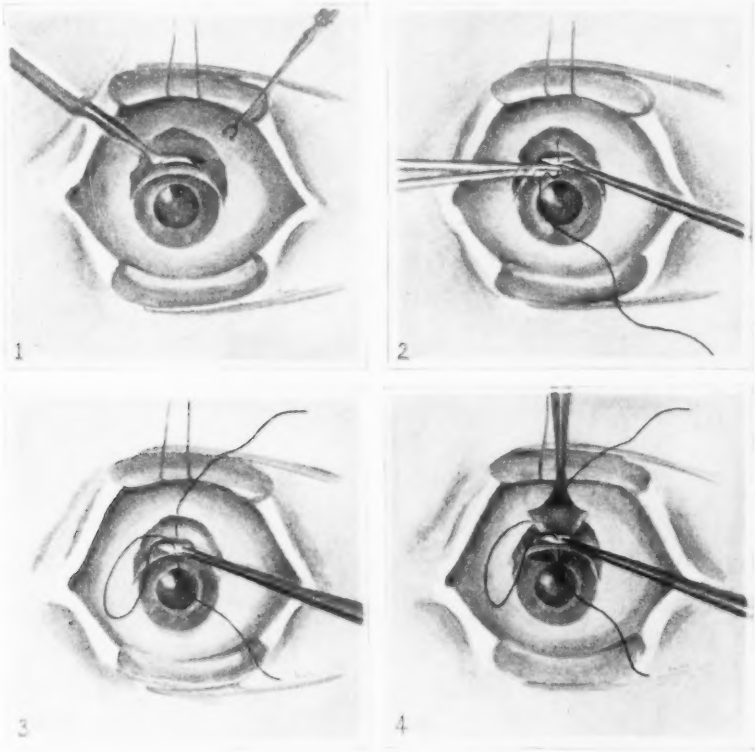
6. Kalt, E.: On the Corneal Suture in Cataract Extraction, Arch. Ophth. **23**:421, 1894.

In order to overcome all these handicaps, and at the same time to afford firm and exact apposition of the lips of the wound, the technic of the scleral flap incision and scleral suture was devised, a description of which follows.

Anesthesia is obtained by retrobulbar injection, and akinesia of the orbicularis oculi muscle, by the O'Brien or Van Lint injection. A retraction suture is placed through the superior rectus muscle at its insertion. This suture is left in place with no traction placed on it and is not used unless it becomes necessary, and then only with utmost care.

knife being kept at a shallow level while the incision is made in order to avoid cutting through the sclera, which in this position is 0.5 mm. thick. A side to side, as well as a slightly rocking, movement of the knife is used in making the incision. The knife is carried toward the limbus and stops short of opening the eyeball (fig. 1).

The size of the scleral flap is more easily controlled with the concave knife than with the Lundsgaard knife. The concave knife forms a concentric, wedge-shaped scleral flap, with its base at the limbus. If the Lundsgaard knife is



Figs. 1-4.—*1* shows the conjunctival flap dissected down over the upper half of the cornea and a concentric, wedge-shaped scleral flap being made by means of the concave scleral knife.

2 shows the suture being applied through the conjunctival flap, the tip of the scleral flap and the edge of the cut scleral surface of the upper lip of the incision.

3 shows the suture in place. The scleral flap has been turned down and the loop drawn aside, demonstrating the interval between the two parts of the scleral suture. The size of the interval depends on the height of the scleral flap.

4 shows how the eyeball is fixed by grasping the scleral flap with a toothless conjunctival forceps. The point of the keratome has been placed in the groove at the base of the scleral flap and has entered the anterior chamber.

A small conjunctival flap of about 3 mm., is dissected over the upper half of the cornea to the limbus. The eyeball is fixed with a scleral pick, and, by means of my scleral knife, which is concave anteriorly, or a Lundsgaard knife, the scleral flap is made. The incision starts 1 to 1.5 mm. above the limbus, the scleral

used, the edges of the flap are incised to the limbus on either side. A fine black silk suture on a sharp, curved atraumatic needle is now passed through the conjunctival flap, the tip of the scleral flap and the corresponding portion of the upper scleral lip and, finally, the edge of the bulbar conjunctiva, above. The loop is drawn

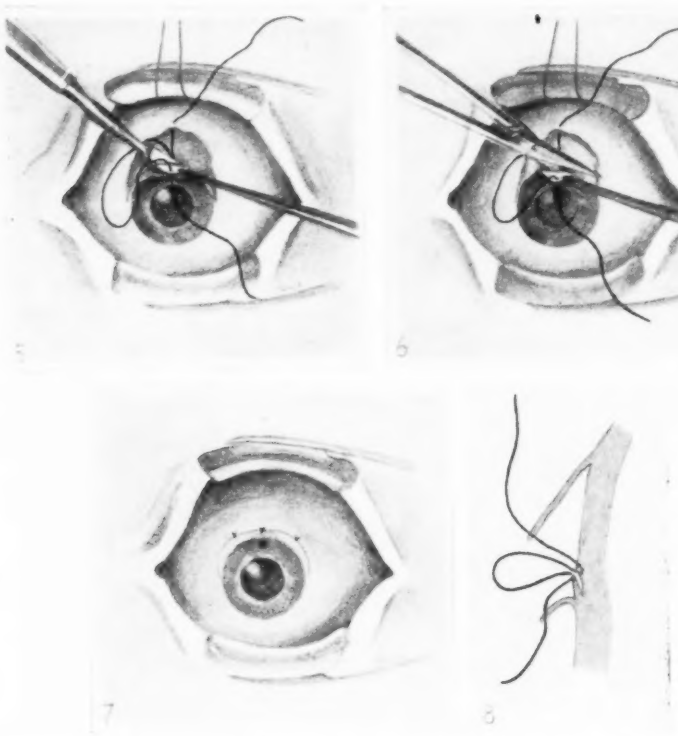
aside to permit the making of the incision (figs. 2 and 3).

When the scleral flap is turned downward toward the cornea, an interval of 1.5 to 2.5 mm. is created between the two parts of the suture, the size depending on the height of the scleral flap. This leaves ample space for the cataract knife to pass, without the risk of cutting the suture. For additional accuracy, however, I prefer to use the keratome and scissors in making the incision. Fixation of the eyeball is secured by grasping the scleral flap with a toothless conjunctival forceps, and the keratome is placed in the groove at the base of the scleral flap (fig. 4). The keratome is made to enter the anterior chamber, and the incision is enlarged with Stevens' scissors (fig. 6).

Should the anterior chamber be very shallow, as in a case of glaucoma combined with cataract, an *ab externo* incision may be made. In such a case one may use a Lundsgaard knife in place of the keratome. With the suture in place, the scleral flap is fixed and held away from the eye. The scleral knife now continues the incision into the anterior chamber (fig. 5). The pressure exerted by the scleral knife is exactly countered by the traction with the forceps on the scleral flap; therefore there should be no prolapse of iris into the wound. The incision is now enlarged with scissors.

If, in the process of making the scleral flap, the anterior chamber is inadvertently opened and a prolapse of the iris occurs, the iris is replaced, and the scleral suture is taken with almost the same ease as with an intact eyeball. However, the upper lip of the scleral wound is fixed with a fine-toothed straight iris forceps in order that no pressure be exerted on the eye while the needle is being passed through the upper scleral lip. The opening in the anterior chamber is enlarged with scissors and the incision completed.

On completion of the extraction, the scleral suture is firmly tied, and two or more auxiliary conjunctival sutures are placed (fig. 7). Figure



Figs. 5-8.—5 illustrates an *ab externo* incision. The Lundsgaard scleral knife, in place of the keratome, is used to enter the anterior chamber.

6 shows enlargement of the cataract incision by means of scissors.

7 shows closure of the wound after the cataract has been extracted. The scleral suture has been tied tightly, and two auxiliary conjunctival sutures have been taken.

8 is a diagrammatic representation of the suture, which has passed through the conjunctival flap, below, the tip of the scleral flap, the upper lip of the sclera and the conjunctiva, above.

8 illustrates the position of the suture through the conjunctiva and the lips of the scleral wound.

SUMMARY

It has been shown that firm and accurate closure of cataract wounds definitely reduces the incidence of postoperative complications and diminishes the amount of postoperative astigmatism. A new method for securing such closure of the cataract wound, namely, the technic of the scleral flap incision and scleral suture, is described. It permits the suture to be taken through the lips of the wound with exactness, and the suture may be tied tightly without buckling or inversion. The scleral flap serves as an excellent point of fixation, and when it is turned downward toward the cornea, there is an ample interval between the parts of the scleral suture to permit the passage of a cataract knife or keratome without risk of severing the suture. I believe that with a little practice the occasional operator should be able readily to master the several steps of the method.

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LAW OF PHYSIOLOGIC AGING AS DERIVED FROM LONG RANGE DATA ON REFRACTION OF THE HUMAN EYE

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FORMER RESULTS AND PROBLEMS

In former papers by one of us (F. B.)[1] and by his collaborators Stainhaus[2] and Heidemann,[3] it has been shown that a statistical correlation exists between the development of presbyopia, i. e., loss of the power of accommodation due to aging, on the one hand, and the span of life, on the other. Statistically speaking this means that persons with early progressing presbyopia die early and that persons with late progressing presbyopia die late. Furthermore, by securing death records in a considerable number of cases (about 1,000) it was clearly demonstrated in the work of Steinhaus[2] that the correlation just mentioned is due primarily to the large subgroup of cases in which "apoplexia" or "heart stroke" (chiefly occlusion of the coronary arteries) was registered as the cause of death.

These former observations are demonstrated in tables 1 and 2.

The life expectancy of men and women who died of apoplexy or "heart stroke" for three classes of presbyopia is shown in table 1.

The column headed "Normal Presbyopia" includes the cases of persons whose presbyopia, measured by the addition for near vision, was found to be equal to the average with a margin of $\pm \frac{1}{8}$ D. All persons with presbyopia of a degree higher than average were classified as having "supernormal presbyopia" and those with presbyopia of a degree lower than normal were said to have "subnormal presbyopia."

The statistical facts presented in tables 1 and 2 indicate that aging in man must follow certain definite physiologic laws. The exact nature of these laws, however, could not be revealed by these statistical studies, which were based on data not giving information on the gradual development of presbyopia in the individual case. Our present paper abandons this statistical approach and deals with the nature of the laws which govern the gradual development of presbyopia in the individual case.

TABLE 1.—*Life Expectation for Three Classes of Presbyopia of Persons Whose Cause of Death Was Arteriosclerosis* **

Age Group	Life Expectancy in Years		
	Supernormal Presbyopia	Normal Presbyopia	Subnormal Presbyopia
40-43	20.7 (33)	26.7 (61)	.....
44-49	17.9 (86)	22.5 (83)	31.8 (54)
50-53	15.5 (51)	18.9 (34)	23.2 (51)
54-58	11.2 (29)	13.9 (26)	19.8 (15)
59-63	11.4 (45)	10.3 (14)	13.4 (23)
64 and over	6.4 (22)	9.9 (41)	10.9 (44)

* The number of cases is shown in parentheses.

TABLE 2.—*Average Life Expectancy for Three Classes of Presbyopia of Persons of All Age Groups with Different Causes of Death (Bernstein 1937)**

Causes of Death	Life Expectancy in Years		
	Supernormal Presbyopia	Normal Presbyopia	Subnormal Presbyopia
Arteriosclerosis.....	15.0 (264)	19.5 (259)	22.2 (237)
Cancer.....	13.0 (47)	11.4 (34)	12.8 (38)
Tuberculosis.....	11.9 (42)	10.8 (35)	10.8 (26)
Grip.....	15.6 (68)	14.3 (40)	13.7 (62)

* The number of cases is shown in parentheses.

NATURE OF THE DATA

The present study is based mainly on data from the histories in 3,000 cases from the files of

This study was made with the support of a grant from the Council for Research in the Social Sciences of Columbia University.

1. Bernstein, F.: (*a*) Alterssichtigkeit und Lebenserwartung, Forsch. u. Fortschr. **8**:272-273, 1932; (*b*) Die natürliche Lebensdauer des Menschen und ihre statistische und individuelle Beurteilung: Comitato italiano per lo stufio dei problemi della popolazione, Rome, Instituto Poligrafico dello Stato Libreria, 1932; (*c*) Biologische Faktoren der Verminderung der Sterblichkeit, ibid., 1932.

2. Steinhaus, H.: Untersuchungen über den Zusammenhang von Presbyopie und Lebensdauer, unter Berücksichtigung der Todesursachen, Arch. f. Augenh. **105**:731-760, 1932.

3. Heidemann, R.: Presbyopie und Lebensdauer, Inaug. Dissert., Göttingen, 1932.

Dr. John E. Weeks, former head of the department of ophthalmology, New York University. These data became available through the courtesy of Dr. Webb Weeks, late head of the department of ophthalmology, New York University. The patients had been under observation for periods ranging from six to forty years, covering the period in which presbyopic changes take place.

In these 3,000 cases, all records were discarded in which the patient showed lower visual acuity. Data in all cases of cataract and, later, of glaucoma were considered only for the earlier, normal, years. All cases were excluded in which refraction seemed to be affected by general disease. Particular care was taken to eliminate all cases of diabetes. Cases of myopia in which there was a tendency to an increase in myopia during the period of observation were also discarded. Even in the category of constant myopia we retained only cases in which the myopia did not exceed — 1.5 D.

It soon became evident that the period of presbyopia to be studied had to be decidedly longer than six years if conclusive quantitative results pertaining to gradual development were to be obtained. Therefore, only the cases were used in which the presbyopic period covered at least twelve years. In addition, this period had to include at least six observations in each individual case.

Of the 3,000 cases collected, 344 satisfied these strict requirements. In each of the observations, the glasses prescribed fully corrected the vision to normal, so that the visual acuity was 20/20 according to the Snellen test. Whenever there was reason to assume a change in the reading distance, a corresponding correction was made, or the data were discarded. The logical continuation of the work by Steinhaus and Heidemann on presbyopia would have been a study of the changes in power of accommodation with age in single subjects. Such a study requires observations made especially for that purpose. Data from prescriptions, on the other hand, are the only ones available in sufficient numbers for a study of the development of refraction in individual subjects, with the necessary extension over long periods.

All three elements of the prescription, namely, the sphere for distance, the astigmatic correction by a plus or a minus cylinder and the correction for near vision, change with age.

The corrections prescribed have two purposes: to enable the patient (1) to read at the distance of 6 meters and (2) to read with ease standardized print at a convenient reading distance.

Since for most patients of presbyopic age the second correction is the more important, and since it includes the elements of refraction studied by Steinhaus and Heidemann, we decided to study the development of refraction with regard to the correction for near vision, with elimination however, of the correction for astigmatism, since astigmatism may develop according to laws of its own.

The total refraction of the reduced eye of Listing (one medium) consists of the sphere which osculates the refracting surface and a hyperboloid surface of Gaussian curvature zero, which is approximated by a crossed cylinder. The osculating sphere is corrected by a sphere whose diopter strength is the sum, y, of the sphere for distance, the addition for near vision and half the diopters of the correcting plus cylinder minus half the diopters of the correcting minus cylinder. The crossed cylinder is corrected by a crossed cylinder, C, with opposite signs of the two curvatures. The crossed cylinder C consists of two crossed cylinders, of which the first has half the strength of the plus cylinder and the second, placed at right angles to the first, has half the strength of the minus cylinder. This separation of the correction remains valid also for the eye with several diffracting media.

This separation of the correction into the sum y and the crossed cylinder C is merely formal. But since Wibaut [4] has shown statistically that the two radiuses of curvature of the cornea change with age in opposite directions exactly the same amount, so that the middle value remains unchanged with age, it is to be assumed that the astigmatism of the cornea is described by a changing crossed cylinder. Apparently the cornea changes with age by being bent in the vertical direction without any kind of deformation [5] so that the increase of curvature in the one principal section is accompanied with an equal decrease of curvature in the other.

Stern and Rosenberg [6] conducted a study of astigmatism in the 3,000 cases on the basis of the ophthalmometric readings and the subjective and objective determinations of astigmatism always made for each new patient. They showed statistically that the axis of the lenticular astig-

4. Wibaut, F.: Biologisch-statistische Refraktionsuntersuchungen, Munich, J. F. Bergmann, 1932.

5. This entails the constancy of the geometric mean of the radii, but since the difference of the radii is small their arithmetical mean will also remain practically constant.

6. Stern, C., and Rosenberg, H.: The Relative Contribution of Cornea and Lens in the Changes of Astigmatism with Age, Thesis, New York University, 1940.

matism remains unchanged with age. This suggests that the process underlying the change of astigmatism of the lens, whenever it occurs, will hardly add to or subtract from the spherical refraction.

One must conclude, therefore, that it is reasonable to expect that the processes underlying the change in the total spherical correction for near vision, y, and the processes underlying the change of the crossed cylinder, C, act so that the first processes change sphere in sphere and that the second processes change crossed cylinder in crossed cylinder. In studying the development of y, we therefore assumed that we had eliminated the influence of all processes causing changes of astigmatism with age. An illustration of this situation could be seen in a case in which the refraction of the two eyes was the same from 40 to 51 years of age but in which astigmatism continued to develop in the right eye while in the left eye a weak astigmatism, such as had existed in equal degrees in the two eyes, remained unchanged. During the whole subsequent period of observation, extending from the age of 51 to that of 73½ years, prescription for the right eye and that for the left differed only by the addition of a crossed cylinder for the right eye. This crossed cylinder was apparently the true expression for the change in astigmatism of the right eye. There were other cases of a similar, but less striking kind, and no case was found which contradicted the theoretic assumption made here.

It will be noted that the processes involved in the development of y are two: first, the senile increase in hypermetropia and, second, the decline of the power of accommodation. It will be seen later that these two processes are closely related and that they can be traced back to a common cause (see "Comment").

It may be stated in advance that for the study of the process of aging the analysis of y offers one great advantage. The value for y undergoes changes in development far past the age of 60, at which age the addition for near vision in usual practice attains a final value. In the data of Dr. Weeks the final value of the addition in most cases is already attained at the earlier age of 55. This constituted an additional reason for concentrating on the study of the total spherical correction, y, for near reading.

GRAPHIC REPRESENTATION OF THE DATA

All data on long range development have been plotted on coordinate paper, as illustrated in case 143 (fig. 1). The symbols used are self explanatory.

We have tried to interpolate graphically and numerically the values for the total correction, y[7] as a function of age through the equation:

$$(1) \quad y = A\ [1 - 2^{-(x - x_0)/c}]$$

This has been done with the aid of semilogarithmic paper, to which the bases 2 in formula 1 refers. The value for y is measured in diopters, and x, in 10 year units. For each eye the value A, called an asymptote, was determined at first by trial and error, A being varied so that the points representing the values for $A - y$ were finally lying close to a straight line on semilogarithmic paper (fig. 2, case 143). This yields an angle, alpha, between the upper part of the

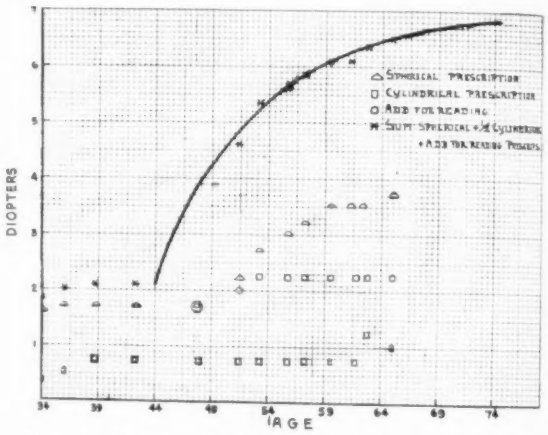

Fig. 1.—Graph showing corrections prescribed for the right eye of a patient (case 143) with presbyopia under treatment over a period of thirty years.

straight line and the x axis to the left of it, the cotangent of which is C. The constant x_0 is the

7. In the corrections the development of which is studied here, the distance of the lens from the eye enters as an unknown, variable quantity. In case this distance is recorded, which was not done in our data, that value should be used to calculate the true refractions and to determine A and c from them. This, of course, still requires an assumption about the distance of the first principal point from the surface of the eye.

In order to illustrate what changes would occur if true refractions were used, the distance of the lens from the surface of the eye is assumed to be 12 mm. and the distance of the principal point from the surface of the eye 1.35 mm., in accordance with Gullstrand. In case 143, for example, we obtained $A = 7.70$ instead of $A = 7$. But c remained wholly unchanged.

In fact, the true refractions are proportional to the corrections belonging to a given distance of the lens from the eye, with a factor of proportionality which is fairly constant within the limits of refractional changes occurring during presbyopia. Therefore the formula 1 with the same c but a different A fits corrections as well as the true refractions in an individual case.

age at which the ordinate of the straight line is equal to A. In the first diagram (fig. 1), y would be zero if the curve could be prolonged backward until it cut the x axis.

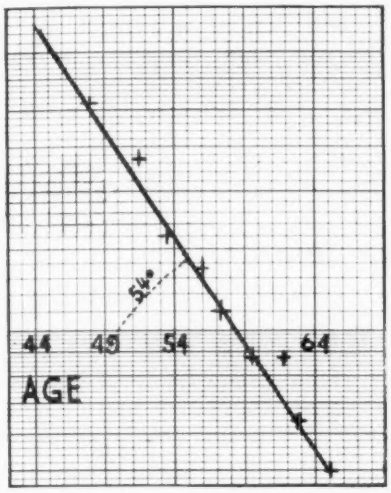

Fig. 2.—Values for $A - y$ (formula 1) for the right eye in case 143, plotted on semilogarithmic paper. The plus sign indicates asymptote A ($= 7$ D.) minus sum y.

In case 143 (fig. 1) it can be observed that up to the age of 42 years y remains almost constant. After that age the correction for distance begins to rise and, at about the same time, the addition for reading does also. The astigmatism remains constant throughout.

The trial and error method of determining A is fairly accurate, but the following numerical method proved finally superior. From any set of values of y given for three successive equidistant values of x, A can be determined by the following rational formula, first developed by J. Lipka:

$$(2) \quad A = y_1 + \frac{(y_2 - y_1)^2}{2y_2 - y_1 - y_3}$$

The application of this formula requires generally the calculation of y_2 through interpolation in order to have three equidistant ordinates. A more general method for three nonequidistant ordinates requires the solution of a trinominal equation with irrational exponents. Such a direct determination of A was worked out, and tables were calculated. This procedure will be published elsewhere.

FIT OF THE FORMULA

The accuracy of determination of the spherical correction is 1/8 D., and in correction for near vision it is seldom more than 1/4 D., so that the accuracy of determination of y lies somewhere between 1/8 and 1/4 D.

Table 3 is so arranged that each point deviating more than 3/8 D. from the corresponding point of the straight line is listed three times—as deviating more than 1/8 D., as deviating more than 1/4 D. and as deviating more than 3/8 D., from the value on the line. As the survey shows, the number of points deviating more than 1/8 D. appears small when the accuracy of the determination of y is taken into account. The number of points deviating more than 1/4 D. is still smaller, and the number of points deviating more than 3/8 D. appears very small and, when checked, is found to be attributable mainly to discontinuous changes in correction for near vision.

TABLE 3.—*Goodness of Fit Sample of 10 Cases* *

Age Interval of Observation, Yr.	Asymptote		Angle Alpha		Age Xo		No. of Points (n — 3)	No. of Observations with Deviations in Diopters of More Than					
								Right			Left		
	Right	Left	Right	Left	Right	Left		1/8	1/4	3/8	1/8	1/4	3/8
40-70½	4.62	4.62	46.5	46.5	47½	37½	7	..	..	..	..	..	..
42-69	4.88	5.25	50	50	42½	42	5	2	1	..	..	..	..
28-59½	5.50	4.88	50.5	50.5	37½	37½	6	2	1	1	..	..	..
39-57	5.00	5.00	55.5	56	36½	36½	7	1	1	..	1	..	..
36-55	5.75	5.75	63	63	41¾	41¾	4	1	1	1	1	1	1
35-64½	5.75	5.25	58	58	38½	38½	5	1	1	..	1	..	..
44-58	6.50	5.75	49	50	38½	38½	5	1	..	..	1	..	..
34-61½	4.50	4.62	51	51	36½	36½	5	2	1	1	..	..	..
38-63	4.75	4.75	56	56	36	36	7	2	..	..	1	..	..
47-67	4.38	4.38	55	55	42½	42½	6	1	..	..	1	..	..

FIRST (GLOBAL) STATISTICAL TEST OF THE EXPONENTIAL LAW OF AGING

A similar test is given in table 4 for 400 eyes. The cases are grouped according to the reduced number of prescriptions ($n - 3$) derived from the individual cases. This table shows that the more observations for a single eye, the more accurate is the determination of the three constants of the curve which expresses the development of the refraction, and therefore the more satisfying is the representation. In fact, in 115 cases with 8 or more observations each, covering a period of twenty-two and eight-tenths years in which the most pertinent check could be made,

only 10 per cent of the observations were found with deviations of more than ¼ D. The deviations in this 10 per cent of cases were mostly attributable to discontinuous changes in the prescription for near vision.

If the index, y, of total refraction develops according to a general aging process, no difference in the trend of aging between the right and the left eye is to be expected. This conclusion, indeed, has been confirmed except only in cases in which the condition was manifestly pathologic.

It sufficed, therefore, in plotting the data in a case to use only one eye, preferably the one with the greater visual acuity. But it was of interest to note that the two eyes, although having equal trends, expressed by c, differed frequently with regard to the asymptotic value of the total refraction.

but

$$(4) \quad A' = A.2^{-(s_0 - x_0)/c}$$

which results from

$$a' = A\ [1 - 2^{-(s_0 - x_0)/c}]$$

a' being the value of the sum y at the time, s_0, age of onset of presbyopic changes. Accordingly, the straight line in the semilogarithmic plane representing equation *1* represents also equation *3*.

Our first test of linearity made use of both eyes of a subject, as though they represented independent observations. The deviations from the straight line in the logarithmic plane are composed of those originating (*a*) from the spherical correction for distance, (*b*) from the cylinder and (*c*) from the addition for near vision. Of these deviations, the last are normally always identical for the two eyes, while the deviations for (*a*) and (*b*), resulting from independent examination, vary independently. The

TABLE 4.—*Goodness of Fit of Curve 1:* $y = A\ [1 - 2^{-(x - x_0)/c}]$

Eyes Grouped According Reduced Number of Points (n − 3)*	Number of Eyes	Reduced Number of Observation Points	Average Age at First Prescription, Yr.	Average Age at Last Prescription, Yr.	Average Duration, Yr.	(*a*) Reduced Number of Observation Points (*b*) Reduced Number of Observation Points, in Percentages With Deviations from Curve 1 Less Than 1/8 D.	Between 1/8 and 1/4 D.	More Than 1/4 D.
1 and 2, inclusive	115	203	44.30	61.04	16.74	(*a*) 122 (*b*) 59.60	(*a*) 45 (*b*) 22.67	(*a*) 36 (*b*) 17.73
3 and 4, inclusive	169	093	41.39	63.78	22.39	(*a*) 309 (*b*) 60.71	(*a*) 133 (*b*) 22.43	(*a*) 100 (*b*) 16.86
5 and above	115	698	42.97	65.77	22.80	(*a*) 484 (*b*) 69.34	(*a*) 131 (*b*) 18.77	(*a*) 83 (*b*) 11.89
Total	399	1,494	43.78	64.33	20.55	(*a*) 966 (*b*) 63.77	(*a*) 309 (*b*) 20.77	(*a*) 216 (*b*) 15.46

* Number of observed points minus 3 points.

This difference as a rule was present at, and even before, the moment when the curve of total refraction started to rise and then remained constant throughout the whole course of the later development. If the amount of total refraction at the age of onset of presbyopia is indicated by a', the formula for total refraction as a function of age x for each eye is then:

$$(3) \quad y = a' + A'\ (1 - 2^{-(x - s_0)/c})$$

where s_0 is the age of onset of the presbyopic change, a' being positive for persons found to be hypermetropic and negative for persons found to be myopic at the time, s_0, of onset of the presbyopic change. Thus, a' is different for the two eyes while s_0, A' and c are the same for the two eyes. As an example, we cite case 58 (figs. 3 and 4). The difference in a' between the two eyes amounts to 1 D.

It is perhaps not superfluous to emphasize that the two equations *1* and *3* are valid at the same time. We have, therefore, not only

$$A = A' + a'$$

values of y belonging to the points on the two straight lines, however, are nearly identical for the two eyes, because both represent the trend of aging in the same subject.

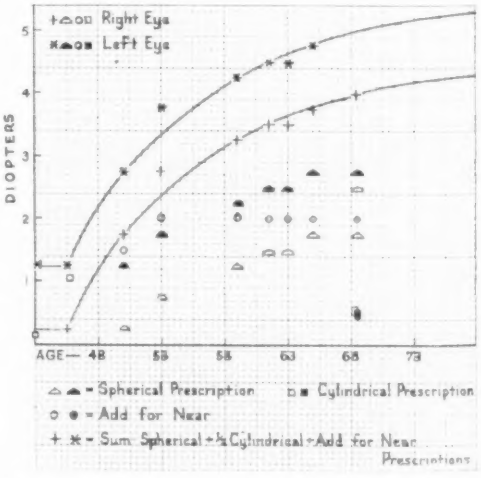
Right Eye
Left Eye
DIOPTERS
AGE— 48 53 58 63 68 73
△ ▲ = Spherical Prescription
□ ■ = Cylindrical Prescription
○ ● = Add for Near
+ ✱ = Sum: Spherical + ½ Cylindrical + Add for Near
Prescriptions

Fig. 3.—Curves for total refraction as a function of age x for each eye in case 58 as expressed by formula 3.

In testing the linearity we first tried to combine the data for the two eyes, using, however, the addition only once, but it proved to be more profitable to use one eye only, preferably the one with the greater visual acuity. An elaborate combination of the data from the two eyes in each case could be dispensed with as long as an increase in the number of cases yielded improvement of the test to any desired accuracy. In this way, a test of linearity of the logarithmic graph, using only independent observations, was made for each eye of 200 subjects. The results of the tests are shown in tables 5 and 6.

TABLE 5.—*Test of Linearity of the Straight Line on Semilogarithmic Paper for 200 Subjects, Using Three Points of the Eye with the Best Visual Acuity*

Diopters, Each Eye	Residual Points
0 - 1/8	506
1/8 - 2/8	153
2/8 - 3/8	29
3/8 and more	16
Total	704

TABLE 6.—*Percentages of Residual Points for the Two Eyes*

	0-1/8 D.	1/8-2/8 D.	2/8-3/8 D.	3/8 D. and More
Eye with best visual acuity	71.97	21.73	4.12	2.27
Other eye	65.03	21.84	7.93	5.20
A tentatively formed weighted average gives percentages:	69.09	21.74	5.72	3.45

The accuracy of determination of distance vision lies between 1/8 and 1/4 D. We believe that the 45 and 704 deviations beyond 2/8 D. are not representative of the actual state of vision. We found that they were due mostly to relatively high changes in the addition. In some cases the deviation seemed to be due simply to contradictory statements of the patients, as judged from the records, since the patients then returned after shorter intervals than usual. In a few other cases the oculist may have had a reason for giving temporarily either more or less corrective glasses. In 37 out of 179 cases the first prescriptions were too strong, and in 4 cases they were too weak, to be in accordance with the linear graph. In such cases the first prescription was not used in determination of the linear graph.

The small number of the deviations beyond 2/8 D. does not indicate in any way chance variation, and therefore we did not apply statistical routine procedure for determination of the best fitting straight line but proceeded graphically, selecting the three points which, according to judgment, seemed the best choice for leading to a straight line representing the total trend. The 200 cases indicated in tables 5 and 6 were those with the relatively greatest number of observations, leading thus to the most significant test of linearity.

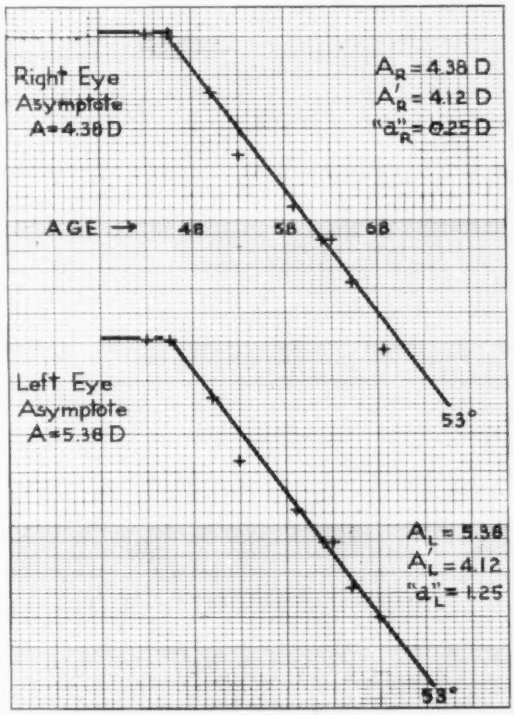

Fig. 4.—Values for $A - y$ for each eye in case 58, plotted on semilogarithmic paper.

But the 143 cases of presbyopia of shorter duration also confirm the law, as seen in table 7.

TABLE 7.—*Test of Linearity of the Straight Line on Semilogarithmic Paper for 143 Subjects, Using Three Points of the Eye with the Best Visual Acuity*

Diopters, Each Eye	Residual Points
0 - 1/8	664
1/8 - 2/8	223
2/8 - 3/8	81
3/8 and more	53
Total	1,021

For 60 cases in which enough values for presbyopia were given for the determination of an asymptote, the average difference between the angle alpha of the straight line, representing the development of the addition for near vision, and the line representing the development of the total index of refraction (sum y) was found to be only 1.008°. The law of linearity is valid for both sexes. Whether or not a sexual difference exists in the constant of the linear law will be discussed later. No change of trend at any par-

ticular age, such as that of the menopause, has been found.

PREDICTION OF LATER DEVELOPMENT OF REFRACTION FROM KNOWLEDGE OF THE PRESCRIPTIONS OF AN EARLIER PERIOD

An interesting application of the law of aging is the possibility of using it for prediction of later development of refraction. For such a study we selected 80 cases in which the age interval from x_0 to about 65 years or more was covered by a sufficient number of points of observation. The index, y, of physiologic aging at the age of 65 for these cases was calculated in two ways.

First, only 4 or 5 of the earliest observations were plotted, and from these the preliminary asymptote was determined graphically on semilogarithmic paper and numerically with the aid of the formula

$$(2) \quad A = y + \frac{(y_2 - y_1)^2}{2y_2 - y_1 - y_3}$$

Thus, the straight line on semilogarithmic paper was drawn, and from it the preliminary angle alpha and the value of the index at the age of 65 were determined by extrapolation, the result constituting the predicted value at the age of 65.

TABLE 8.—*Prediction of Physiologic Age at 65 Years from First Four or Five Observations (10 Selected Cases)*

Case No.		Asymptote, D.	Angle Alpha, Degrees	Index y at 65 Yr., D.	Age at First Observation	Age at Fourth (or Fifth) Observation	Age at Last Observation
286	Predicted	3.88	49	0.45	42¾	50½	66
	Observed	4.00	51	0.44			
326	Predicted	4.25	52	0.45	42¾	47¾	65
	Observed	4.38	51.5	0.47			
304	Predicted	7.00	51.5	0.33	37	46¾	62½
	Observed	7.00	51.5	0.33			
318	Predicted	2.88	47	0.48	42	56	68½
	Observed	2.88	47	0.48			
327	Predicted	8.00	51	0.80	43	52¾	62½
	Observed	7.62	56	0.55			
339	Predicted	4.75	54	0.57	46	56¼	69
	Observed	4.62	52	0.62			
341	Predicted	4.88	49	0.50	42	52	66¼
	Observed	4.62	50.5	0.43			
337	Predicted	4.62	52.5	0.60	45	55	73
	Observed	4.50	55	0.50			
289	Predicted	6.38	47	0.60	45	58	66½
	Observed	6.50	48	0.61			
292	Predicted	5.62	47	0.46	37	53¼	63¼
	Observed	5.50	48.5	0.43			

Second, the value of the index at the age of 65, if not given by direct observation, was determined by interpolation between or extrapolation from the nearest observations, the result constituting the "observed" value of the index at the age of 65.

Third, the angle alpha and the asymptote were always determined on the basis of all observations in order to obtain what we consider the true values of the angle alpha and the asymptote. The second determination, of course, includes the data of a case used in the first, but with comparatively small influence, in view of the long period of the observation.

TABLE 9.—*Prediction of Physiologic Age at 65 Years from a Partial Period of the First Four or Five Observations (80 Cases)*

No. of Cases	Average Duration of Partial Period, Yr.	Average Difference Between Predicted and Observed Values: Asymptote A, D.	Angle Alpha, Degrees	Index y at 65 Yr. of Age, D.
20	5.75	0.131	2.925	0.100
20	8.55	0.119	2.925	0.070
20	10.45	0.103	2.735	0.115
20	14.0	0.094	1.300	0.071
Total 80	9.68	0.127	2.288	0.089

A comparison of the predicted index and the observed index, of the preliminary asymptote and the true asymptote and of the preliminary alpha and the true alpha angle show close agreement. The agreement between the "preliminary" and the "true" values is, at the same time, also an agreement between prediction and observation. We have tabulated the results of such a prediction for 10 cases chosen at random (table 8) and for the total series of 80 cases (table 9).

As the tables show, there was an average difference of only 0.127 D. between the two values of the asymptote, an average difference of 2.288° between the two values of the angle alpha and an average difference of only 0.089 D. between the predicted and the observed index at the age of 65.

THEORETIC CONSIDERATIONS

The simple law equation (1) for the development of y calls for casual explanation. Equation 1 may be written in the form

$$(5) \quad A - y = A\,2^{-(x - x_0)/c}$$

The left side of formula 5 is the increase of the addition for near vision plus the increase of the true sphere for distance (corrected so that the astigmatism is eliminated).

As has already been mentioned, the relation between the two components of y varies with the amount of atropinization. Herrenheiser[8] advanced the hypothesis that the increase in senile hypermetropia is due to the gradual manifestation of undetected preexisting hypermetropia with increasing age. His theory is in agreement with modern experience as to the action of drugs

8. Herrenheiser, J.: Die Refraktionsentwicklung des menschlichen Auges, Ztschr. f. Heilk. **23**:342-377, 1892

in samples of organisms. It has been shown that there exists such a wide variation of effect that there will be found in every large sample some organisms not affected by the drug. Straub and Falkenburg [9] found 6 cases in which atropine did not reveal hypermetropia in a sample of 59 subjects, all of whom were in the twenties and were known to have been hypermetropic at an earlier age. The authors concluded that tectonic changes must have taken place. But in the light of recent pharmacologic experience such a conclusion does not seem well founded. Similarly, the conclusion that the increase in senile hypermetropia must be explained by tectonic changes because of the failure of atropine to reveal a preexisting hypermetropia, as correctly postulated by Herrenheiser, seems, again, logically unwarranted.

In addition, it is to be noted that the smooth muscles easily assume a state of semipermanent contraction, which, according to Bethe,[10] can be sustained with little expenditure of energy. A semipermanent contraction, as described by Bethe, may not be sensitive to atropine at all. If this theory is accepted, the two components of $A - y$ are both manifesting the power of the ciliary muscle to contract, and they constitute together the true power of accommodation at the age x. It has, then, to be explained further only why this power decreases exponentially.

The theory of accommodation proposed by Helmholtz in the form in which it is usually stated is not suited as a basis of explanation. The failure of this theory to account for the acceleration of the effect of atropine with age has been noted already by Duane.[11] The theory, however, as far as the kinematics of the mechanism is concerned, has been amply proved by Hess [12] and, recently, by Fincham.[13] Fincham presented proof that the capsule of the lens is the source of the capillary forces which round out the lens in accommodation, while the interior of the lens consists of inert matter in a semifluid state.

As Duane pointed out correctly, in the usual statement of the theory, the ciliaris muscle spends an increasingly smaller part of its power in releasing the capillary action of the capsule of the lens, so that, contrary to experience, atropine should paralyze the needed effort to a progressively less degree. The equation 5, as well as the experiments of Duane, requires that the ciliaris muscle spend its full power in the act of maximal accommodation, as postulated in the theory of Tscherning. We have, therefore, modified the theory of accommodation proposed by Helmholtz in the following way:

The power of the ciliaris muscle is increasingly absorbed in the action of contracting against the elastic resistance of the zonula-sclera system, which holds the lens in suspension. That the zonula is connected with the sclera by connective tissue, which, by-passing the ciliary body, can transmit the tension, is anatomically certain. The elastic resistance, $R(x)$, is assumed to increase according to an exponential law, and this explains the equation 5. It explains, also, why the effect of atropine is accelerated with age when the power of the ciliaris muscle becomes largely absorbed by its action against the increasing elastic resistance, $R(x)$.

That the increase of $R(x)$ with age is exponential conforms to the laws of colloid chemistry. The tissues of the body are colloid solutions, which desiccate with age in proportion to the water still present. This leads to the exponential law of decrease of water content of the tissues and, consequently, to the exponential increase of the constant, $R(x)$, of Hooke's law of elastic resistance, considered as a function of the age x. Measurements of the dry weight of various parts of the body have been made by Buerger and Schlomka (Bernstein [1b]). They conform to a law of exponential increase with age.

In the foregoing explanation, the implicit assumption has been made that the nearly maximal contraction of the ciliaris muscle, assumed and sustained in accommodation for reading, decreases in proportion to the increase of the elastic resistance, $R(x)$, of the zonula-sclera system. Hill and Hartree confirmed the previous observations of Fick that in series of isometric contractions of striated muscle the length varies approximately in inverse proportion to the maximal tension, although there are some deviations from proportionality at either end of the scale. In the presbyopic period, hypertrophy of the ciliary muscle can be disregarded and the supposed inverse proportionality between tension and length can be assumed, in analogy to the

9. Straub, M., and Falkenburg, J.: Ueber die normale Refraktion des Auges und die Hypertropie bei angeborener Amblyopie, Arch. f. Augenh. **26**:336-362, 1892.

10. Bethe, A.: Die Dauerverkürzung der Muskeln, Arch. f. d. ges. Physiol. **142**:291, 1911.

11. Duane, A.: Studies in Monocular and Binocular Accommodation, Am. J. Ophth. **5**:865-877, 1922; Accommodation, Arch. Ophth. **5**:1-14 (Jan.) 1931.

12. Hess, C.: Die Refraktion und Akkommodation des menschlichen Auges und ihre Anomalien, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, W. Engelmann, 1910.

13. Fincham, E. F.: The Mechanism of Accommodation, Brit. J. Ophth., 1937, supp. 8, pp. 5-80.

relationship observed in a series of isometric contractions of one and the same muscle.

Further, we have assumed that the two step contraction of the ciliary muscle, namely, first from the resting stage to semipermanent equilibrium and then from that equilibrium to nearly maximal contraction, is dynamically equivalent to a one step contraction. This is approximately true, since, as Duane has observed, patients after atropinization may have lost for several months a small amount of their power of accommodation, so that the near point recedes somewhat, but they will still be able to accommodate nearly as much as before.

CONCLUSION

It has been the object of this paper to establish and explain the law of aging as revealed by the long range development of refraction.

This goal has been reached by elimination of questions interesting in themselves, such as the development of astigmatism and the respective developments of distance refraction and accommodation for near vision. For the establishment of the physiologic law as such, questions like a possible sex difference in aging may be disregarded and reserved for special studies.

The most interesting aspect of the result gained is that a method has been achieved by which one may determine fairly accurately a constant, c, which describes the exponential velocity of the change with age of the total spherical refraction of a subject.

In the biologic interpretation of the law, this velocity, c, is nothing else than the exponential velocity with which one tissue of the body changes from the sol stage to the gel stage. No assurance can be given that the other tissues of the body change with the same exponential velocity. But it is plausible that a correlation exists between the velocities of desiccation of the various tissues of the body with age. The results of Steinhaus and Heidemann with regard to a correlation between presbyopia and death from natural causes must be understood as based on a correlation between the velocities of desiccation of the various tissues of the body. The length of life is conditioned not only by internal but by external factors. In view of the large variation of the external factors of environment, this correlation must be rather great in order to express itself so clearly in the cited statistics.

It cannot be denied that considerable interest and prospect of basic clarification attaches to a study of such correlations in a direct manner, for which the way now seems to be opened. If it is desired to use immediately the results here established for the purpose of general medicine, the way in which it can be done is evident.

The values of c derived from the 344 cases of presbyopia of long duration vary considerably. These values of c, or, better, the values of the angle alpha of the logarithmic diagram, can be recorded in different classes, say, five classes, in which the values for the angle alpha determined from the history of refraction can be recorded for each subject. This classification record, then, tells whether the person is aging normally, whether he is aging more rapidly than normal or whether he is aging more slowly than normal. With much greater reliability than the classifications of Steinhaus and Heidemann, such a classification would characterize the individual patient as an aging organism.

Although we have prepared such a table, we do not include them in this paper, for the reason that the 344 cases, selected on the basis of long duration, represent a biased sample. It is evident that such bias excludes the cases of presbyopia of short duration and, in consequence, the cases of quick and unfavorable aging. Our norm would lie at too high a level. We should classify too many cases in the most unfavorable category.

This deficiency of our classification might be corrected after we have learned the date of death of the 344 persons, most of whom have already died. The determination whether, with the evidence now at hand, and with a plausible allowance for the bias of our table, the diagnosis of the velocity of aging, c, from the history of refraction as presented here, may add to the knowledge of a basic age characteristic of an individual subject, with sufficient reliability to be of unmistakable significance for general medicine, is at present still in the experimental stage of trial and error.

SUMMARY

As reported in earlier publications by one of us (F. B.) and his collaborators Heidemann and Steinhaus, a statistical correlation exists between presbyopia, as measured by the gradual loss of power of accommodation, and the length of life. By securing death records in about 1,000 cases, Steinhaus showed that the correlation was due exclusively to the cases in which death was the result of arteriosclerotic changes. In 5,000 cases obtained from various sources, Steinhaus and Heidemann found no difference between men and women in the average amount of developing presbyopia. Neither was there any difference between the urban and the rural population or among different social classes.

The present study is summarized.

1. From the files of Dr. John E. Weeks, former head of the department of ophthalmology of New York University, 3,000 cases were selected, in which the development of refraction and the changes in prescriptions could be followed through periods ranging from six years to over forty years, covering ages from 35 to the highest calendar ages. From these 3,000 cases were selected 344 in which presbyopia had been present more than twelve years and 6 or more prescriptions had been given. In none of these cases were there any pathologic traits or more than 1.5 D. of myopia, and in all visual acuity was 20/20 in the Snellen test.

The refraction in each case, consisting of the spherical prescription for distance, the cylinder prescription and the additional spherical prescription for reading distance was plotted against age.

2. The total refraction was represented by the sum of the following two independent optical components: (*a*) a sphere of the amount of the sum, *y*, of the spherical prescription for distance, the addition for near vision and half the strength of the plus cylinder prescribed for astigmatic correction; and (*b*) a crossed cylinder, *C*, the positive component of which was half the positive cylinder of the astigmatic prescription, with the same axis.

The laws of development for *y* and *C* are wholly independent of each other. Comparison of the results for the two showed that in individual cases the crossed cylinder, *C*, may develop differently for the right eye and the left eye, while the total spherical correction, *y*, may develop identically for the two eyes.

3. An attempt was made to represent the development of the sphere *y* by a curve according to the formula:

$$(1) \quad y = A\ (1 - 2^{-(x - x_0)/c})$$

where *x* is the calendar age and *A*, x_0 and *c* are constants, determination of which requires the knowledge of three values of *y*, calculated for three different ages. The determination was first made with the aid of semilogarithmic paper, for which 2 is the base of the logarithmic system. The constant *A* was first determined by trial and error. Later, the following formula was used:

$$(2) \quad A = y_1 + \frac{(y_2 - y_1)^2}{2y_2 - y_1 - y_3}$$

which is valid if the three ages, x_1, x_2, x_3, for which the y_1, y_2, y_3 are calculated, follow each other at equal intervals. It is usually necessary to gain the y_2 for the middle value by interpolation.

Tables for the determination of the constants for the more general case in which *y* is calculated for three ages not following each other at equal intervals have been calculated.

4. For the test of goodness of fit of the formula (1), 1,494 observations, not used in the determination of the three constants, were available for 400 eyes belonging to the 200 persons with the maximum number of observations.

Of these 1,494 observations, 69.3 per cent were lying within the ⅛ D. distance of the straight line of the semilogarithmic graph and 88.1 per cent within the ¼ D. distance. For 144 eyes with a lesser number of observations and a shorter period, 84.5 per cent of observations were lying within the ¼ D. distance of the straight line.

Because of the equality for the two eyes of the addition prescribed for near vision, a statistical dependence is created between the deviations used in the test. In the second test, in which this statistical dependence was avoided by using the data for one eye only, 72 per cent were found within the ⅛ D. distance and 93.7 per cent within the ¼ D. distance for the 200 subjects.

5. An individual analysis of the relatively few cases represented by the 6.3 per cent deviations of more than ¼ D. distance led to the conclusion that each of these deviations could be attributed either to no change in the prescription after a certain lapse of time or to considerable changes in the addition for near vision. The smaller deviations of less than ¼ D. distance from the continuous curve (1) are caused by the lack of intervals finer than ⅛ D. in the lenses of the trial case, which are graded in ⅛ D. steps for the spheres and cylinders.

6. In the cases in which the two eyes differed during the period of observation by a constant sphere, it was more convenient to use instead of equation 1, a formula with four constants, one of which expresses this constant difference. This formula is

$$(3) \quad y = a' + A'\ (1 - 2^{-(x - s_0)/c})$$

where a', the prepresbyopic value of *y* at the age s_0, may differ in the two eyes. a', s_0 and *c* are identical for the two eyes.

The values of *y* calculated from formulas 1 and 3 do not differ for values of *x* greater than s_0.

For 60 cases the development of the addition for near vision could be studied together with the development of *y*, and both could be interpolated by the formula 1 and graphically represented by straight lines in the logarithmic diagram. The angle alpha, the cotangent of which is *c* of formula 1, differed in the same case on the average only 0.008°, so that in these cases the trend in *y*

and the trend in the decline of the power of accommodation proved to be essentially the same.

7. The 80 cases of presbyopia of longest duration and maximum number of observations were used for prediction of the total refraction at the age of 65 from the knowledge of 4 or 5 prescriptions between the ages 40 and 50 years. At the age of 65, there was an average difference of 0.089 D. between the predicted and the observed value of y.

8. It is concluded that the explanation of the exponential law requires (*a*) the adoption of the theory of senile hypermetropia, proposed by Herrenheiser, which, to the exclusion of the postulation of tectonic changes, explains senile hypermetropia as the manifestation of preexisting hypermetropia temporarily compensated by a contraction of the ciliary muscle; and (*b*) a change in the theory of accommodation proposed by Helmholtz with regard to the dynamic aspect of the theory, namely, that the decrease of accommodation with age is due to the increased resistance to the action of the ciliary muscle caused by the aging of the tissues of the zonula and the sclera.

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ACCOMMODATION OF PRESBYOPIA AND ITS CORRECTION

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The correct addition for near vision in presbyopia depends primarily on the amount of available accommodation and secondarily on the selection of the best fixed distance for the near work.

In correcting presbyopia, it has been customary among ophthalmologists to give the patient only one test, that is, to determine the addition with which the patient "reads best" or feels most comfortable at the desired distance on the Prince rule. In ophthalmology one has at least three common methods of determining the correct distance refraction, or punctum remotum: (1) retinoscopic examination with cycloplegia, (2) trial case determinations with cycloplegia and (3) the postcycloplegia test. I recommend three methods for measuring the near point refraction, punctum proximum, or accommodation. I believe that the near point in the refraction of presbyopia is as important as the far point. Each of my three tests depends on distinctly different types of thresholds of visual sensation. There is no doubt of the statistical value of the measuring of samples in groups of three.

RESERVE ACCOMMODATION

The reserve accommodation in presbyopia is that part of the total accommodation which must be set aside for comfort in doing ordinary continuous close work. For example, a patient with total accommodation of 1.50 D. with an addition of a 2.00 D. lens reads best at 40 cm. (2.50 D. on Prince's rule). The extra 1.00 D. is the reserve accommodation. In this paper, I use a basic reading distance of 40 cm. (2.50 D.) as a standard in all my calculations. I shall show that exactly 1.00 D. is the normal reserve accommodation which must be used in all presbyopic corrections. Hence, the total accommodation less + 1.00 D. is the available accommodation. By subtracting the patient's total accommodation from 3.50 D., one obtains the indicated addition for this basic distance of 40 cm.

THREE METHODS OF MEASURING TOTAL ACCOMMODATION IN PRESBYOPIA

TEST 1: *The Near Blur Test.*—With the patient's distance correction in place, determine his total accommodation by Duane's method [1] or with my one-two line test.[2]

The one-two line test is as follows: With the distance correction before the right eye and with the left eye occluded, place a + 1 to a + 3 D. spherical lens so that the near point will be about 25 cm. from the eye. On one side and at the end of a small white card, place a fine black line, similar to Duane's black line on a white card. On the other side or opposite end of this card, place two similar parallel black lines, separated by an interval equal to the width of either line (figure, *A*). Present the parallel lines before the right eye much too close to be seen distinctly. Move these two parallel lines away from the eye until the patient sees two lines. Note the reading on the Prince rule. Then, bring the single line in until the patient announces that it is "slightly blurred," "worse" or "not so good." Repeat this process until the difference of the two readings is 2 cm. or less. If there is still doubt as to the correct measurement of accommodation, place the fine print (figure, *B*) close to the patient's eyes. Tell him to read it aloud as soon as he can. Note the point on the rule where he begins to read. In measuring the accommodation, always record the blurring of the line coming in as diopters on the Prince rule and not as centimeters. There is a distinct advantage in thinking of the amounts of accommodation in terms of "diopters required" distance rather than of linear measurement.

TEST 2: *The "Reads Best" Test.*—Place the indicated addition in front of the patient's distance correction and ask him where he "reads best" the fine print on the test chart. It is convenient to have this chart rectangular and mounted on a handle (figure, *B*). This should be read at 40 cm. if the measurement of accommodation is correct. Hence, to test his accommodation in this manner, ask him where he reads this fine print best on the Prince rule with the indicated addition in the trial frame. This point

1. Duane, A.: A Standard Test-Object for Determining the Near Point and Range of Accommodation, Ophth. Rec. **18**:358-360 (July) 1909.

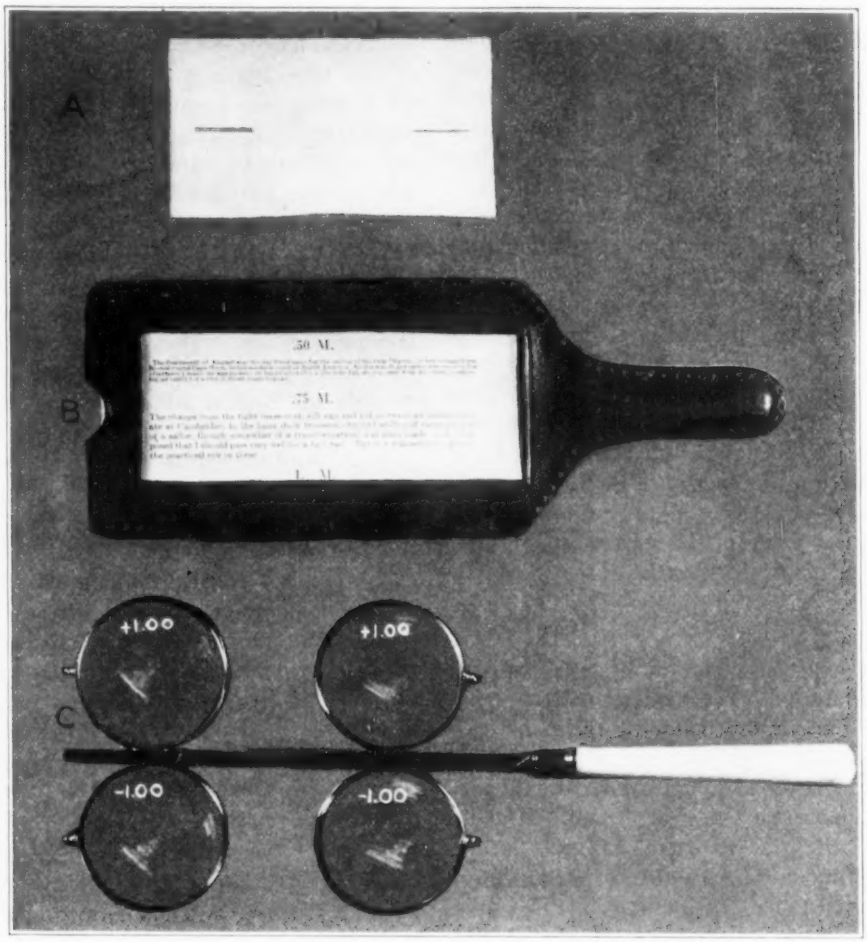
2. Slataper, F. J.: Total and Reserve Accommodation in Presbyopia, Texas State J. Med. **21**:536-540 (Jan.) 1926.

expressed as diopters minus the addition plus 1.00 D. is his total accommodation.

TEST 3: *The + and — 1.00 D. Spheres Readability Test.*—Now with + 1.00 D. spheres (figure, *C*) added to the correction for distance previously determined and the indicated addition, place the reading chart beyond 50 cm.; slowly bring the chart toward the patient's eye and ask him to tell where he can read some of the letters of a word. I use a small, paddle-shaped frame to hold the reading chart. This device enables the patient to place the small reading matter directly over the markings on the Prince rule. This chart becomes partly readable at or near 40 cm. if the accommodation and the addition are correct. Record this "working far blur" accommodation in diopters. This "readability" or "blur" accommodation less 1.00 D. is the patient's actual "far partly readable," or "far blur" accommodation.

The "far readability" or "far blur" accommodation can be determined with the + 1.00 D. spheres addition in two ways: (1) Bring the print in from 50 cm. until it is "partly readable," or (2) push the print out from about 25 cm. until it is slightly blurred. Deduct 1.00 D. to get the actual far point accommodation.

Place a — 1.00 D. sphere binocularly before each eye in place of the + 1.00 D. spheres, as in the preceding test, and hold the chart close to the patient's eyes. Ask him to tell when he can read a few letters of a word of the fine print, which should become partly readable at 40 cm. (2.50 D.). This reading plus 1.00 D. is his actual "near partly readable" or "near blur" accommodation, expressed in diopters. The "near readability" or "near blur" accommodation with — 1.00 D. spheres can be measured in two ways: (1) Push the print out from about 25 cm. until it becomes "partly readable," or (2) bring the print in from 50 cm. until it becomes "slightly blurred." The average near and far blur readings should be at 40 cm. (2.50 D.), if the accommodation and addition are correct.



A, accommodation card; *B*, reading chart in handle, and *C*, + and — 1.00 D. spheres on handle.

In this third test of the total accommodation, the "partly readable" procedure is more dependable than the "slightly blurred" determination because the ophthalmologist can check the patient's cooperation by hearing him read the words aloud. The "average near and far readability" accommodation less the addition plus 1.00 D. is equal to his total accommodation. In case the patient has less than 5/5 vision, he should be examined for his "reads best" and his "average readability" accommodation at his "optimum" reading distance and not at 40 cm.; e. g., a patient with a vision of 5/10 should be tested at 33 cm. (table 5). The average "near and far readability" test with + and — 1.00 D. spheres is the most accurate test of accommodation in cases of presbyopia.

Comment.—The "reads best" test and the + and — 1.00 D. spheres readability test can be used only in cases of presbyopia (3.50 D. or less of accommodation). However, the one-two line test can be used for measuring any amount of accommodation if the patient has sufficient intelligence.

TESTS OF COOPERATION

In making the preceding tests for the near point, if the results seem contradictory, the patient should be tested for his cooperation. There are three methods which I use for doing this.

1. *The Near Blur Cooperation Test.*—Add or subtract a 1.00 D. sphere from the addition in the trial frame. If his near blur accommodation does not change 1.00 D. on the rule, he is not cooperating.

2. *"Reads Best" Cooperation Test.*—Use a pair each of + and — 0.50 D. spheres on a handle. Add + 0.50 D. spheres to the addition for the near point and the correction for distance. His "reads best" accommodation should be 0.50 D. nearer on the Prince rule than before. Now, reverse the procedure. Add — 0.50 spheres to the correction. The "reads best" accommodation will be 0.50 D. less on the rule. This test is a quick method of proving the patient's cooperation. If the patient fails to change the point on the Prince rule by 0.50 D., he is not cooperating.

3. *The + and — 1.00 D. Spheres "Partly Readable" Average Cooperation Test.*—Change the addition in the trial frame 0.50 or 1.00 D. Then find the average "far partly readable" accommodation with + and — 1.00 D. spheres. The average should be exactly the amount by which the addition in the trial frame was changed.

Comment.—When one finds a patient who does not cooperate in one of the aforementioned tests, ignore that test and use the other two. Do not allow the patient to know that he is not cooperating. Tell him that the patient is always right and that all of the mistakes are yours. Many of the patients who say they are ten years younger than their real age are also guilty of not cooperating in one of these cooperation tests. I have never seen a patient who could fool me in more than one of the tests because each of the three methods depends on a different type of threshold of visual sensation.

HISTORY OF THE THREE ACCOMMODATION TESTS

Soon after I began the private practice of ophthalmology, I became convinced that the correct addition for presbyopia should be based on the patient's accommodation and not on his alleged age. From the beginning, I have measured the accommodation in diopters by a near blur test (Duane's method or my one-two line test) in all cases of refraction except those of young children and aphakic persons. After three years of practice, I stated:

> The amount of sphere added for near in an ordinary case of presbyopia depends upon three factors: the working distance, the total accommodation, and the reserve accommodation.[3]

In that study of normal reserve accommodation,[3] I made some mistakes, which were due to wrong "assumptions." One such assumption was that the average working distance of all presbyopic corrections was 33 cm., instead of 40 cm., which is more nearly correct. Again, the assumption of an "average addition of 3.00 D. for a patient of 60 years with normal vision" was not correct. In that paper, the reserve for 2.50 D. accommodation or less varied from 1.21 to 1.00 D. instead of being exactly 1.00 D.

When, in September 1935, I began the individual tests of the first series (200 cases), I was ignorant of the relation of the average blur test to the "reads best" and the "near blur" accommodation test. In that series, I found that the "average near and far blur" test was a practical test for accommodation.

In the second series of tests (100 cases), begun in January 1940, I made the following observation: 1. With the aid of the + and — 1.00 D. spheres, the "average near and far blur" accommodation could be measured better at 40 cm. than without them at 66 and 28 cm. This eliminated the confusion of the patient between sharpness of focus and magnification and minification

3. Slataper, F. J.: The Normal Reserve Accommodation in Presbyopia, Arch. Ophth. **55**:370-376 (July) 1926.

of the test object. 2. The "blur amplitude" was 2.00 D. Having become aware of these two facts, I began the third series of tests.

About November 1941, I began the routine of taking the average near and far blur readings with the + and — 1.00 D. spheres in the fitting of glasses for presbyopia as a check on the "reads best" and the "indicated addition" determination based on the near blur (Duane's or my one-two line) test for the total accommodation. In the individual cases of the third series (300 cases) the measurements of average readability or average blur accommodation were all taken with the + and — 1.00 D. spheres to prevent the confusion of the test objects by their being magnified or minified.

THEORETIC CONSIDERATIONS

The presbyopic eye corrected for distance and for near vision in getting a sharp retinal picture is similar to a camera being focused for a close-up photograph and follows some of the same laws of optics.

DEFINITIONS

The point on a Prince rule at which the patient "reads best" corresponds to the best focus of the camera.

In the eye, the "near blur" point on the rule represents the nearest area of the picture sufficiently in focus to be partly recognizable.

In the eye, the "far blur" point represents the part of the picture farthest from the anterior focus of the eye where print is partly recognizable. The near and the far blur point are always measured in diopters from the eye.

In the eye, the area between the near and the far blur point is called the blur amplitude.

In the camera, the area between these two points is called the depth of focus.

The near blur point is obtained by bringing fine print or a line in toward the eye until it blurs or by pushing print or two lines out away from the eye until it is partly readable.

The far blur point is obtained by pushing print out until it blurs or by bringing it in from a distance until it is partly readable.

The near and far blur points are practically the same, respectively, as the near and far "partly readable" points. The slight difference of measurements by these methods is that of the reaction times of the patient and of the ophthalmologist. This is always made less with practice.

DEDUCTIONS

1. *Algebraic Demonstration of Blur Amplitude.*—In the corrected presbyopic eye, as in a camera, the sum of the near and far blur points divided by 2 is always equal to the "reads best" point or "best focus" when measured in diopters from the anterior focus of the eye, as demonstrated by the following formula:

A well known formula[4] for measuring depth of focus as distance in photography is as follows:

$$\left.\frac{\text{F. B. distance} \times \text{N. B. distance}}{\text{F. B. distance} + \text{N. B. distance}}\right\} \times 2 = \text{best focus, or Y}$$

To convert distance in meters into diopters, divide unity by the distance. Substituting diopters for distance in this formula

$$\frac{\frac{1}{\text{F. B.}} \times \frac{1}{\text{N. B.}} \times 2}{\frac{1}{\text{F. B.}} + \frac{1}{\text{N. B.}}} = \frac{1}{\text{Y}} \quad \therefore \quad \frac{\frac{2}{\text{F. B.} \times \text{N. B.}}}{\frac{\text{N. B.} + \text{F. B.}}{\text{F. B.} \times \text{N. B.}}} = \frac{1}{\text{Y}}$$

Inverting the terms of the divisor and multiplying

$$\frac{2}{\text{F. B.} \times \text{N. B.}} \times \frac{\text{N. B.} \times \text{F. B.}}{\text{F. B.} + \text{N. B.}} = \frac{2}{\text{F. B.} + \text{N. B.}} = \frac{1}{\text{Y}}$$

$$\frac{2}{\text{F. B.} + \text{N. B.}} = \frac{2}{2\text{Y}}$$

In equal fractions, if the numerators are equal, the denominators are equal.

Therefore $\text{F. B.} + \text{N. B.} = 2\,\text{Y}$

or $$\frac{\text{F. B.} + \text{N. B.}}{2} = \text{Y}$$

expressed as diopters.

Therefore, the average blur point equals the best focus in terms of diopters. Hence, as in photography, the average of the near and the far blur point, when expressed as diopters, is equal to the best focus. That is, the "reads best" point is equal to the "average near and far blur" point in a presbyopic correction for near vision.

2. *Normal Reserve Accommodation.*—In my three series of cases, I proved that the normal reserve accommodation of presbyopia is exactly 1.00 D. This means that the near blur accommodation is 1.00 D. less than the "reads best" accommodation, since

$$\frac{\text{F. B.} + \text{N. B.}}{2} = \text{Y}$$

as proved, or $\text{N. B.} + \text{F. B.} = 2\,\text{Y}$

4. Stubbs, S. G. B.: The Modern Encyclopedia of Photography, Boston, American Photographic Publishing Company, 1940, vol. 1, p. 523.

Since the near blur accommodation is the normal reserve accommodation and always is equal to 1.00 D., by substituting Y — 1 for N. B.

$$\text{F. B.} + \text{Y} - 1 = 2\,\text{Y}$$
$$\therefore \text{F. B.} = \text{Y} + 1$$

Now, since F. B. = Y + 1 and N. B. = Y — 1, the difference in the value for N. B. and that for F. B. is 2.00 D.

Hence, the far blur accommodation is equal to the "reads best" accommodation plus 1.00 D. Therefore, the depth of focus, or blur amplitude, in presbyopia is 2.00 D., and the near blur is always equal to the far blur.

3. *Physiologic Demonstration of Blur Amplitude.*—This principle that "the average blur" accommodation is always equal to the "reads best" accommodation can be demonstrated with a patient under a cycloplegic after the examiner has finished his refraction at 5 meters. Add + 2.50 D. spheres to his correction for distance. He will read with this best at 36 cm. (2.75 D.). His far blur accommodation will be 2.75 D., with + 1.00 D. added to his correction, and his near blur accommodation, 2.75 D., with — 1.00 D. spheres added in place of the + 1.00 D. spheres. Subtracting and adding 1.00 D. gives his far blur point at 1.75 D. and his near blur point at 3.75 D.; that is, 1.75 D. plus 3.75 D. divided by 2 equals 2.75 D. He reads with a + 2.50 D. addition best at 36 cm. (2.75 D.) because his distance refraction was determined at 5 meters (0.20 D.) instead of at infinity.

In two series of patients, 10 presbyopes and 10 nonpresbyopes given refraction under homatropine cyclopegia, this observation of a difference of 2.00 D. was confirmed for each of the 20 patients. Patients under atropine cyclopegia were given similar tests, with the same results.

This blur amplitude (near blur minus far blur reading) is always 2.00 D., as proved physiologically in the preceding paragraph and by the laws of optics shown in section 2.

4. *Analysis of Blur Amplitude.*—This 2.00 D. of blur amplitude consists of two parts: (1) the true depth of focus, 0.58 D., and (2) the nonparalyzable accommodation of 1.42 D. Ogle [5] has shown that the human eye has an average depth of focus of 0.5 to 0.66 + D. (average 0.58 D.). "The mean of all in table 2 was 0.566." [5]

What is responsible for this 1.42 D. power of accommodation found during cycloplegia and in the presbyope wearing a correction for near vision? It must be due to changes in the position or shape of the lens, because in cases of aphakia I find that the blur amplitude varies from 0.5 to 0.66 + D., in agreement with Dr. Ogle, and not 2.00 D. It cannot be due to the intraocular ciliary muscles, which are not paralyzed by a cycloplegic, because in any presbyope exactly the same blur amplitude of 2.00 D. occurs without cycloplegia as with complete cycloplegia. Could it be due to the extraocular muscles pressing on the vitreous, causing lenticonus, or to a displacement of the lens along the axis of the eye about 0.568 mm. backward for the far blur accommodation and 0.568 mm. forward for the near blur accommodation? According to the lenticonus–vitreous pressure theory a negative pressure would be required for the far blur accommodation. This is impossible on account of the collapsible walls of the sclera. The displacement theory appears the more plausible; that is, the shifting of the lens backward or forward, as in the fishes, of about 0.568 mm. in each direction accounts for 1.42 D. of accommodation.

The mechanism of accommodation consists of two types: (*a*) Change in the shape of the lens. This is abolished by a cyclopegic and ceases with advanced presbyopia. (*b*) Shifting of the lens forward or backward. This causes most of the 1.00 D. change for near or far vision, which is not affected by cycloplegia or presbyopia and must be used as the normal reserve in the fitting of presbyopia corrections for near vision. Reading at the near blur point for long periods quickly becomes tiresome. Reading at the far blur point causes the eyes to have the sensation of "drawing" and may become painful.

5. *Changes in the Visual Angle.*—Many patients confuse sharpness of focus with magnification. The height or tangent of the tall letters of Thorington's + 0.50 D. type on a reading test card is about 2 mm., or 17 minutes of angle at 40 cm. When viewed for comparison, a letter of the same height shows an experimental visual angle of 10 minutes at 66.6 cm. and of 24 minutes at 28.5 cm. These are the distances of the presbyope's far and near blur accommodation when fitted with a glass for near work at 40 cm.

In measurement of the blur accommodation at the far point, 66.66 cm., and at the near point, 28.57 cm., the same letters occupy 2.4 times as large a visual angle at the near blur as at the far blur point. This type of magnification, a larger retinal picture, makes the test object appear more readable, while the actual focus may not be so good. If the near and the far blur accommodation are tested each at 40 cm., this confusion of magnification, minification and sharp-

5. Ogle, K. N.: Measurements on the Depth of Focus of the Human Eye, with Various Test Objects, Masters Thesis, Dartmouth College, Hanover, N. H., 1927.

ness of focus can be eliminated. Since the blur amplitude was exactly 2.00 D., an addition of + 1.00 D. for the far blur and . — 1.00 D. for the near blur accommodation would be ideal. Hence, to test these blur or readability accommodations at 40 cm., add to the selected presbyopic correction — 1.00 D. spheres for the near and + 1.00 D. spheres for the far point. I used a pair of + and a pair of — 1.00 spheres on a handle for this purpose (figure, *C*). The increased accuracy in these tests for near and far blur accommodation in my third series of cases was ample proof of the usefulness of the method.

6. *Accommodation Formulas.*—Since the normal reserve accommodation in correcting presbyopia is 1.00 D., 3.50 D. less the total accommodation is the standard addition for reading at 40 cm.

My formulas for total accommodation threshold tests are as follows:

1. The near blur test (my one-two line test or Duane's test): The near blur accommodation less the addition equals the total accommodation.

2. The "reads best" test: The "reads best" accommodation less the addition + 1.00 D. equals the total accommodation.

3. The + and — 1.00 D. spheres test (average readability or blur test): The average readability or blur accommodation less the addition plus 1.00 D. equals the total accommodation.

4. Far blur or far readability test: The far blur or the far readability accommodation less the addition plus 2.00 D. equals the total accommodation.

Duane's test is the one most often described by writers on accommodation. The "reads best" principle is most often used in prescribing lenses for near vision, but it is not recognized as an actual test of total accommodation. However, in presbyopia, it is the second most accurate single method of measuring total accommodation. The average blur test with the + and — 1.00 D. spheres is the most accurate accommodation test because it depends on two separate thresholds of sensation, which are easily compared at the same distance. This observation was confirmed by results in my third series of cases. The far blur test is the least accurate single test because it is more difficult for the patient to comprehend and cooperate.

In my hands, the near blur method is always used as a monocular test; the "reads best" and the + and — 1.00 D. spheres (average readability or blur) method are always binocular tests in the determination of a presbyotic correction. I always make the three tests in the order indicated.

NORMAL RESERVE ACCOMMODATION

The average total accommodation in each case was determined by taking a third of the sum of total accommodation obtained by the three methods: (1) my one-two line procedure, (2) the "reads best" method and (3) the + and — 1.00 D. spheres average blur test.

$$\text{Average total accommodation} = \frac{\text{1-2 line acc.} + \text{"reads best" acc.} + \text{avg. blur acc.}}{3}$$

The reserve accommodation in each of the 600 cases was figured from the average accommodation. This average accommodation was figured by two methods in the first series, of 200 cases, and by three methods in the second series, of 100 cases, and in the third series, of 300 cases. The reserve was figured according to the following formula: Average accommodation plus the addition minus 2.50 D., the standard distance accommodation, equals the normal reserve accommodation.

$$\text{Average accommodation} + \text{addition} - 2.50\text{ D.} = \text{normal reserve accommodation}$$

STATISTICS ON NORMAL RESERVE ACCOMMODATION (Tables 1 and 2)

In table 1, the normal reserve accommodation for the additions of 1.00 to 2.50 D., inclusive, are shown as averages in 600 cases. As my work became more accurate in the succeeding series, the normal reserve accommodation approached closer to 1.00 D. The mean deviation, the standard deviation and the probable error consistently became less. This is shown in table 2. Thus, in the last series one could say that the normal reserve accommodation is exactly 1.00 D.

In a similar manner, I made a small series of tests with additions from 2.75 to 3.50 D. and found the normal reserve accommodation to be exactly 1.00 D.

RELATIVE ACCURACY OF THE THREE ACCOMMODATION TESTS (Tables 3 and 4)

In the third series, of 300 cases, the one-two line accommodation test was found to be the most frequently wrong. It showed an error of 16.33 + per cent. The "reads best" accommodation test was wrong in 2.33 + per cent of the cases. The + and — 1.00 D. spheres readability accommodation test was the most accurate, as it showed an error of 0.33 + per cent.

The fact that the tests are always made in the order indicated tends to make the last test the most accurate. However, this factor will not account for the great difference in their accuracy.

A glance at table 4, presenting a summary of the average values for the three accommodation tests, will show the relative accuracy in this series. For the "reads best" and the + and — 1.00 D. spheres average blur accommodation test the mean deviation, the standard deviation and the probable error are much less in the third series than in the first and second series. The more I worked with accommodation, the less reliable I found the near blur test (my one-two line test and Duane's test).

TABLE 1.—*Statistics on Normal Reserve Accommodation*

Addition, D.	Normal Reserve Accommodation	Mean Deviation	Standard Deviation	P. E.
First Series (300 Cases)				
1.00	0.98	0.05	0.52	0.351
1.25	1.02	0.07	0.75	0.506
1.50	0.98	0.04	0.46	0.310
1.75	0.99	0.05	0.61	0.411
2.00	0.99	0.05	0.65	0.438
2.25	1.00	0.04	0.57	0.384
2.50	1.02	0.04	0.46	0.310
Total.....	6.98	0.34	4.02	2.710
Average.....	0.997	0.048	0.574	0.387
Second Series (100 Cases)				
1.00	0.992	0.035	0.051	0.034
1.25	0.981	0.052	0.080	0.054
1.50	1.008	0.031	0.075	0.030
1.75	0.965	0.042	0.046	0.031
2.00	1.002	0.023	0.042	0.028
2.25	0.994	0.025	0.044	0.030
2.50	1.025	0.032	0.035	0.024
Total.....	6.967	0.240	0.373	0.231
Average.....	0.995	0.034	0.053	0.033
Third Series (300 Cases)				
1.00	1.000	0.008	0.023	0.0157
1.25	1.002	0.007	0.016	0.0110
1.50	1.002	0.003	0.007	0.0052
1.75	0.994	0.009	0.015	0.0102
2.00	1.001	0.002	0.006	0.0043
2.25	1.002	0.021	0.002	0.0013
2.50	1.010	0.009	0.024	0.0168
Total.....	7.011	0.059	0.093	0.0645
Average.....	1.001	0.008	0.013	0.0092

TABLE 2.—*Summary of Data in Table 1*

Series	Average Reserve Accommodation	Mean Deviation	Standard Deviation	Probable Error
First.....	0.997	0.048	0.574	0.387
Second.....	0.995	0.034	0.053	0.033
Third.....	1.001	0.008	0.013	0.0092

BEST FIXED DISTANCE

After the patient's total accommodation is decided on, the next step is to determine his proper reading distance.

The best fixed distance for near work depends on the following factors:

Visual Acuity.—The best reading distance is most easily determined by the visual acuity, as shown by table 5. This table is based on visual efficiency, as shown by Snell [6] and Snell and Sterling [7] and used by Lebensohn [8] in his "near vision test charts." It is assumed that 40 cm. is the average normal reading distance for 20/20 vision. Hence, the best reading distance for any visual acuity is obtained by multiplying the visual efficiency of that acuity by 40 cm.

With a visual acuity of 20/200 or 20/100, reading is impossible except for short periods of time. A + 13.00 D. lens held in the hand is a more practical glass for near vision than when it is placed in a spectacle. A + 4.00 D. addition is about as strong an addition in a bifocal as can be used. This often forms a very useful glass for a patient with vision of 20/70. A patient with 20/40 vision should never be fitted for any distance beyond 33 cm. from his eye. I know several presbyopic bookkeepers with this vision who are very efficient in their work.

If the patient has unequal vision, the average "diopters required" distance of each eye should be used, provided that the difference is 1.00 D. or less (table 5). If the difference is more than 1.00 D., then the patient should be fitted as for "monocular vision" in the better eye. The following examples may be given:

Vision of 20/60 in the right eye requiring 3.50 D. and vision of 20/20 in the left eye requiring 2.50 D. gives a difference of 1.00 D. The sum of 2.50 D. and 3.50 D. divided by 2 equals 3.00 D., the average diopters of correction required. The patient should be fitted for 33 cm.

Again, vision of 20/70 in the right eye requiring 4.00 D. and vision of 20/16 in the left eye requiring 2.25 D. gives a difference of 1.75 D. The patient should be fitted as for monocular vision. Now, 2.25 D. plus 0.25 D. equals 2.50 D., at a distance of 40 cm. Bifocals of the same strength as the glass for the better eye are optional.

The patient should not be given unequal additions for unequal vision, because each eye requires the same amount of normal reserve accommodation. Unequal additions should be given only for unequal total accommodation, which is rare.

6. Snell, A. C.: Visual Efficiency of Various Degrees of Subnormal Visual Acuity, J. A. M. A. **85:** 1367-1373 (Oct. 31) 1925.

7. Snell, A. C., and Sterling, S.: Percentage Evaluation of Macular Vision, Arch. Ophth. **54:**443-461 (Sept.) 1925.

8. Lebensohn, J. E.: Scientific and Practical Considerations Involved in the Near-Vision Test with Presentation of a Practical and Informative Near-Vision Chart, Am. J. Ophth. **19:**110-117 (Feb.) 1936.

Muscular Balance.—With poor convergence, the patient should be fitted for as far a point as is consistent with reading efficiency. A patient with an excessive convergence should be fitted from 0.25 to 0.50 D. closer than a patient with normal muscle balance. If he has no fusion at the near point, he should be fitted for monocular vision. Bifocals of unequal additions may be found convenient in some cases of external and internal strabismus with fair vision in each eye.

Nature of Occupation.—Many occupations require a special glass for the office, to be used only at work. The patient has an extra pair of general utility glasses which he uses most of the time. As examples: The stenographer with 20/16 vision using a Line-a-time copy holder most of the day should be fitted for 50 cm. The pianist should be fitted for 57 or 60 cm. Both these occupations require extra high, large bifocals. The linotype operator needs the bifocals of the same shape.

In many occupations the bifocal segment must be very low and very small, as in the case of a railroad conductor, truck driver, head janitor or rifleman.

TABLE 3.—*Summary of Data on the Three Accommodation Tests*

Addition, D.	Average Accommodation	"1-2 Line" Accommodation: Total Accommodation	"1-2 Line" Accommodation: Mean Deviation	"1-2 Line" Accommodation: Standard Deviation	"1-2 Line" Accommodation: P. E.	"Reads Best" Accommodation: Total Accommodation	"Reads Best" Accommodation: Mean Deviation	"Reads Best" Accommodation: Standard Deviation	"Reads Best" Accommodation: P. E.	Average Blur Accommodation: Total Accommodation	Average Blur Accommodation: Mean Deviation	Average Blur Accommodation: Standard Deviation	Average Blur Accommodation: P. E.
First Series (200 Cases)													
1.00	2.48	2.49	0.07	0.12	0.081	2.51	0.08	0.097	0.065	2.45	0.08	0.11	0.074
1.25	2.27	2.30	0.17	0.18	0.121	2.28	0.08	0.098	0.066	2.24	0.10	0.13	0.088
1.50	1.98	1.98	0.05	0.071	0.048	2.00	0.06	0.098	0.067	1.96	0.08	0.098	0.067
1.75	1.74	1.74	0.07	0.087	0.059	1.76	0.05	0.065	0.044	1.74	0.08	0.10	0.067
2.00	1.49	1.49	0.05	0.069	0.046	1.52	0.06	0.084	0.057	1.49	0.07	0.087	0.059
2.25	1.25	1.23	0.06	0.084	0.057	1.29	0.072	0.09	0.061	1.21	0.01	0.12	0.081
2.50	1.02	1.00	0.039	0.046	0.031	1.03	0.053	0.083	0.056	1.02	0.089	0.11	0.074
Total			0.509	0.657	0.443		0.455	0.615	0.416		0.509	0.755	0.510
Average			0.072	0.093	0.063		0.065	0.088	0.059		0.072	0.107	0.072
Second Series (100 Cases)													
1.00	2.49	2.48	0.05	0.09	0.061	2.50	0.03	0.052	0.035	2.50	0.02	0.042	0.028
1.25	2.23	2.25	0.06	0.11	0.074	2.24	0.02	0.044	0.030	2.20	0.05	0.10	0.067
1.50	2.01	2.00	0.05	0.084	0.057	2.00	0.03	0.046	0.031	2.00	0.03	0.046	0.031
1.75	1.71	1.62	0.11	0.17	0.115	1.77	0.05	0.077	0.052	1.75	0.07	0.098	0.066
2.00	1.50	1.51	0.04	0.082	0.055	1.50	0.02	0.041	0.028	1.50	0.02	0.038	0.026
2.25	1.24	1.19	0.06	0.12	0.081	1.26	0.04	0.086	0.058	1.28	0.04	0.096	0.065
2.50	1.02	1.02	0.03	0.058	0.039	1.06	0.06	0.10	0.067	0.96	0.09	0.19	0.128
Total			0.400	0.714	0.482		0.250	0.446	0.301		0.320	0.610	0.411
Average			0.057	0.102	0.069		0.035	0.063	0.043		0.045	0.087	0.059
Third Series (300 Cases)													
1.00	2.500	2.500	0.013	0.042	0.0283	2.508	0.013	0.040	0.0270	2.496	0.009	0.033	0.0222
1.25	2.250	2.338	0.091	0.143	0.0964	2.261	0.021	0.051	0.0344	2.240	0.010	0.024	0.0162
1.50	2.002	1.940	0.081	0.187	0.1261	2.005	0.003	0.018	0.0121	2.000	0.001	0.008	0.0064
1.75	1.744	1.716	0.091	0.177	0.1193	1.738	0.008	0.028	0.0191	1.746	0.005	0.014	0.0095
2.00	1.501	1.473	0.115	0.268	0.1809	1.504	0.003	0.014	0.0095	1.500	0.001	0.006	0.0042
2.25	1.252	1.246	0.049	0.118	0.0795	1.258	0.006	0.030	0.0202	1.250	0.003	0.014	0.0094
2.50	1.011	1.010	0.018	0.050	0.0337	1.024	0.016	0.045	0.0303	1.006	0.009	0.024	0.0162
Total			0.458	0.985	0.6642		0.070	0.226	0.1526		0.038	0.123	0.0831
Average			0.065	0.147	0.0949		0.010	0.032	0.0218		0.005	0.017	0.0119

TABLE 4.—*Summary of the Averages of the Three Accommodation Tests (Table 3)*

Series	"1-2 Line" Accommodation: Mean Deviation	"1-2 Line" Accommodation: Standard Deviation	"1-2 Line" Accommodation: P. E.	"Reads Best" Accommodation: Mean Deviation	"Reads Best" Accommodation: Standard Deviation	"Reads Best" Accommodation: P. E.	Average Blur Accommodation: Mean Deviation	Average Blur Accommodation: Standard Deviation	Average Blur Accommodation: P. E.
First	0.072	0.093	0.063	0.065	0.088	0.059	0.072	0.107	0.072
Second	0.057	0.102	0.069	0.035	0.063	0.043	0.045	0.087	0.059
Third	0.065	0.147	0.0949	0.010	0.032	0.0218	0.005	0.017	0.0119

TABLE 5.—*Optimum Presbyopic Reading Distances Based on Visual Acuity*

Acuity		Diopters Required	Distance, Cm.	Visual Efficiency, %
5/61	20/200	12.50	8.0	30
5/30	20/100	5.00	19.6	48.9
5/21	20/70	4.00	25.5	63.8
5/15	20/60	3.50	28.0	69.9
5/12	20/50	3.25	30.6	76.5
5/10	20/40	3.00	33.4	83.6
5/7.5	20/30	2.75	36.6	91.4
5/6	20/25	2.62	38.2	95.6
5/5	20/20	2.50	40.0	100
5/4	20/16	2.25	44.0	110

Length of Arms.—The person with long arms may be fitted differently from the person with very short arms.

Monocular Vision.—A person with monocular vision should have an addition of + 0.25 D. more than the rule.

Duane [9] has shown that in cases of presbyopia the binocular accommodation on an average is 0.25 D. greater than the monocular accommodation. He suggested: "We must reckon with this fact in giving him a reading glass, which must, as a rule, be at least 0.50 D. stronger than if he were two-eyed."

Old Reading Habits.—Many myopes with high astigmatism have acquired the habit of reading 30 to 36 cm. from their eye by taking off their glasses. These patients may be fitted with their bifocals to read at this distance to keep them happy. The same principle applies to a patient who has long worn an overcorrection for near work.

Many presbyopes have never worn a correction for near work until they need a very strong addition. These patients are more comfortable if they are fitted for 44 cm. rather than 40 cm.

9. Duane, A.: Monocular and Binocular Accommodation, Tr. Am. Ophth. Soc. **20**:132-157, 1922.

CONCLUSIONS

1. The correct estimation of the patient's total accommodation is the most important factor in the correction of presbyopia with a glass for near work.

2. It is necessary to use three types of tests to measure accurately the total accommodation in many patients.

(*a*) The near blur test — after Duane.

(*b*) The "reads best" test — the most common method.

(*c*) The + and — 1.00 D. spheres average readability test.

One must continue with these three tests in the order indicated until the readings are all made at the same point on the Prince rule. This procedure seldom fails.

(*d*) The + and — 0.50 D. spheres cooperation test will show the cause in case of failure.

3. The normal reserve accommodation for all near work is exactly 1.00 D.

4. The available accommodation for near work is the total accommodation less 1.00 D.

5. The best fixed distance for close work depends on the following factors: (*a*) visual acuity, (*b*) nature of occupation, (*c*) muscle balance, (*d*) length of arms, (*e*) monocular or binocular vision and (*f*) long-established reading habits.

1110-1111 Medical Arts Building.

REMOVAL OF THE DISLOCATED LENS UNDER A CONJUNCTIVAL POCKET FLAP

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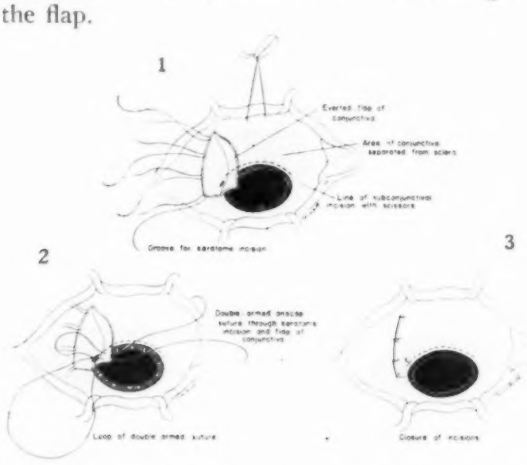
Utilizing ideas from several operators and adding some of my own, I have developed and used in 10 cases a technic emphasizing the following features:

1. A vertical incision of the conjunctiva, about 13 mm. long, beginning 5 mm. from the limbus, at 9 o'clock in the left eye and at 3 o'clock in the right eye.

2. Wide separation of the conjunctiva over the upper half of the globe.

3. Opening of the anterior chamber, with a keratome incision, under the everted edge of the flap.


1
Everted flap of conjunctiva
Area of conjunctiva separated from sclera
Line of subconjunctival incision with scissors
Groove for keratome incision
2
Double armed silk suture through keratome incision and flap of conjunctiva
Loop of double armed suture
3
Closure of incision

Steps in the formation of a conjunctival pocket flap for removal of the dislocated lens.

4. Completion of the section, with scissors, under the flap.

5. Extraction of the lens with the loop, depressing the sclera under the flap.

6. A water-tight closure of the incision.

Control of the eye is obtained with the usual akinesis, retrobulbar injection and superior rectus traction suture. The iris is widely dilated with a short-acting mydriatic. No iridectomy is done. Contraction is maintained for several days with physostigmine.

A groove is made under the everted lower edge of the flap with a Bard-Parker no. 15 blade at the 2 o'clock position in the left eye and the 10 o'clock position in the right eye, and a double-armed silk suture blade secured to an eyeless needle is placed through the cornea at half its depth, the needles pointing in opposite directions. The loop is drawn out, with the threads held apart, and the keratome enters the bottom of the groove, between the two bites. These needles are afterward pulled through the overlying flap, and the suture is tied outside.

Four single-armed silk sutures are placed through the lateral lip of the conjunctival incision, including a small bite of the episclera, and are turned aside, prior to making the section. After extraction of the lens, these sutures are passed through the other edge of the flap and tied, completing the closure.

I have found the most difficult detail to be the placing of the suture through half the layers of the cornea and the correct placing of the keratome incision. I believe, however, that this step is of the utmost importance.

I have had the following complications:

1. Once the suture was caught and divided by the point of the keratome. The suture was reapplied, after the section, without difficulty.

2. Once the iris was drawn up, resulting in elevation of the pupil. This was relieved by iridotomy below.

There have been up to this time no retinal separations.

I have been surprised at the small loss of vitreous. My only explanation is that the positions of the conjunctival and corneal incisions do not coincide. I consider this flap considerably safer than the conjunctival bridge, and I present the method as a fairly safe procedure for the operator of average skill, with the expectation that the ingenious ones will improve on it.

Before submitting this article, I searched the literature for a description of a similar method, and the nearest I could find was one by Czermak [1] of a similar flap made below the equator and one by Tood (1915) of an undermining flap, formed with a knife incision. Neither of these procedures, in my opinion, has all the advantages of the method I have described.

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1. Cited by Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1907, vol. 2.

FOSTER KENNEDY SYNDROME WITH POST-TRAUMATIC ARACHNOIDITIS OF OPTIC CHIASM AND BASE OF FRONTAL LOBES

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The syndrome of atrophy of the optic nerve of one eye and papilledema in the other eye has come to be regarded as evidence of a tumor lying at the base of the frontal lobe. This relation was first pointed out by Paton [1] (1909) and was further elaborated on by Foster Kennedy [2] (1911). Recent studies have indicated that the syndrome may be found at times in association with arteriosclerosis of the internal carotid arteries, which compress the optic nerves and chiasm.[3] Recognition of this observation has tended to indicate that the Foster Kennedy syndrome is found at times in conjunction with disorders other than tumor and has led to elaboration of the conditions under which the syndrome may occur. Experience with a recent case indicates that it may develop also as a result of arachnoiditis in the region of the optic nerves and optic chiasm.

REPORT OF A CASE

History.—S. S., a man aged 42, was admitted to the neurologic service of the Jefferson Medical College Hospital on Oct. 24, 1944 with the complaints of blurred vision and occipital headache. Two weeks before admission he had been struck on the back of the head and rendered unconscious. He was taken to the accident ward of another hospital and discharged after observation, but he continued to complain of occipital headache and appeared mentally dull after the accident. He exhibited progressive mental torpidity and continued to complain of headache. For this reason he was admitted to the hospital for study.

Examination.—Physical examination revealed good development. The patient showed a recent memory defect, in which retention and recall were impaired. Olfaction was preserved. The pupils were equal in size and shape, and both reacted well to light and in accommodation. Examination of the fundi revealed pallor of the left optic disk with blurring of the nasal edge and 3 D. of edema of the right optic disk. There were weakness of the right side of the face of central type, slight weakness of the right hand grip and pronounced unsteadiness in gait. The remainder of the neurologic examination revealed nothing significant.

Repeated examinations of the visual fields showed generalized constriction of the peripheral fields of vision, more advanced in the left eye. There was a central scotoma on the left side. The right blindspot was enlarged. Uncorrected visual acuity was 20/200 in the left eye and 20/30 in the right eye. Roentgenograms of the skull revealed the presence of a linear fracture in the right occipital bone extending from the region of the internal occipital protuberance through the floor of the right cerebellar fossa. The sella turcica was normal, as were the optic foramens. Lumbar puncture yielded clear, colorless spinal fluid under an initial pressure of 320 mm. of water. The total protein content of the spinal fluid was 44 mg. per hundred cubic centimeters. The Wassermann and Kahn reactions of the blood and of the spinal fluid were negative. The electroencephalographic tracings revealed no abnormality in either the resting record or during hyperventilation.

Course.—On the basis of the neurologic and laboratory observations, it was felt that the patient had a brain tumor. Because of the Foster Kennedy syndrome, a basofrontal lesion (meningioma of the anterior fossa) seemed most probable. The absence of olfactory disturbances and the incoordination of gait were disturbing, and a ventriculographic examination seemed indicated. The ventriculograms showed that both lateral ventricles were tremendously dilated symmetrically, with no midline shift and that no air was visible in the third ventricle. The latter observation pointed to the possibility of a tumor in the anterior portion of the third ventricle. Combined encephalographic and ventriculographic examination was attempted but was unsuccessful.

Visual acuity became progressively decreased. About three weeks after his entrance into the hospital the uncorrected visual acuity was 20/200 in the right eye, while the left eye was blind. The Foster Kennedy syndrome persisted. In view of the rapid visual loss and the ventriculographic evidence of a tumor of the third ventricle, exploration was made on November 26, one month after the patient's entrance into the hospital and six weeks after the injury. A transfrontal craniotomy on the left side was performed. Over the entire dura covering the left frontal lobe was a thin hemorrhagic membrane, evidently the result of a hemorrhage of less than six months' duration. As the frontal lobe on that side was examined, it was found firmly attached at its anterior end to the dura by a band of adhesions roughly 1½ inches (3.8 cm.) in length and ½ inch

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1. Paton, L.: A Clinical Study of Optic Neuritis in Its Relationship to Intracranial Tumors, Brain **32**: 68, 1909.

2. Kennedy, F.: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, Am. J. M. Sc. **141**:355, 1911.

3. Yaskin, H. E., and Schlezinger, N. S.: Foster Kennedy Syndrome Associated with Non-Neoplastic Intracranial Conditions, Arch. Ophth. **28**:704 (Oct.) 1942.

(1.27 cm.) in width. In fact, the dura appeared as though there had been an old underlying fracture at this point, which was approximately above the left orbital roof. As the frontal lobe was lifted, more adhesions were found over the left optic nerve. Around the optic nerve they were so dense that it was almost impossible to uncover the nerve. As operative dissection continued, adhesions were found to fill the entire cisterna chiasmatis. The optic chiasm was tightly adherent to the brain and could not be entirely dissected free of it. The dense matting of adhesions gave the gross appearance of a tumor lying just above the chiasm. Investigation, however, revealed that no tumor was present. So obscured were the structures by the dense adhesions that it was difficult to ascertain the normal anatomic markings in this area. In order not to overlook a second lesion in the third ventricle, as indicated by the absence of air filling this region, this structure was next explored through the corpus callosum. Through this incision the third ventricle could be seen to be wide open. The aqueduct of Sylvius could easily be visualized. A small catheter was threaded through the aqueduct for a distance of 3 inches (8.5 cm.) without meeting an obstruction, ruling out a lesion of this structure or of the fourth venricle.

Exploration established the presence of dense basilar arachnoiditis, which was secondary to the cranial trauma. The trauma also explained the dense adhesions between the frontal lobe and the dura and the organized subdural hemorrhagic membrane, which was less than 1 mm. in thickness. A portion of the dura and the associated membrane was removed for study and proved microscopically to be an area of partially organized hematoma. The patient reacted poorly and died ten days after operation.

This patient had a typical Foster Kennedy syndrome which was found to be the result of a dense arachnoiditis extending over the optic chiasm and optic nerves and the base of the frontal lobes. No tumor was found at operation, and the dilatation of the ventricles must be attributed to interference with the circulation of the spinal fluid by the adherent arachnoid, since exploration revealed a patent third ventricle, iter and fourth ventricle. The arachnoiditis was unquestionable and of a degree capable of producing chiasmic symptoms.

COMMENT

Experience[4] has established the localizing value of the Foster Kennedy syndrome in cases of tumor of the anterior fossa. That it may be associated with conditions well removed from the anterior fossa is not so clearly recognized. It has been found in conjunction with a meningioma arising from the right side of the falx (Custodis[5]) and, in another instance, with a tumor of the cerebellum in which the dilated third ventricle was held responsible for the syndrome (Cusick[6]). These exceptions are rare and seem to emphasize that in almost all instances a Foster Kennedy syndrome is found to be associated with a lesion of the anterior fossa in the region of the optic chiasm. To the localization of the tumor in the latter region there are also exceptions, since the syndrome has been observed with tumors of the sphenoid ridge in the parasellar region (Alpers and Groff[7]). In these instances, however, the probability is that the tumors extended forward into the anterior fossa.

The cause of the Foster Kennedy syndrome is in most instances a meningioma. Among other causes which have been listed are tumors and abscesses of the frontal lobes, gliomas of the intracranial portion of the optic nerve and aneurysm of the internal carotid artery (Marchesani[8]). Kennedy[9] described the syndrome in case of aneurysm of the right internal carotid artery. It may be produced also by sclerosis of the internal carotid arteries.

Yaskin and Schlezinger[3] reported 2 cases in which the syndrome was produced by compression of the optic nerves by sclerotic internal carotid arteries. They indicated that the presence of a binasal field defect in association with the Foster Kennedy syndrome necessitated a consideration of sclerotic internal carotid or anterior cerebral arteries as the cause of the syndrome. Schloffer[10] reported a case which is comparable to the 2 cases mentioned. His patient had a Foster Kennedy syndrome with some constriction of the nasal fields of vision. A diagnosis of neoplasm of the anterior fossa was made. At operation a sclerotic right internal carotid artery was discovered compressing the right optic nerve. Necropsy later revealed pronounced atherosclerosis of the vessels at the base of the brain, chiefly the carotid arteries. The right internal carotid artery was so large that the compressed optic nerve was translucent. Changes

4. Cushing, H.: Macewen Memorial Lecture on Meningiomas Arising from the Olfactory Groove, Lancet **1**:1329, 1927. Schmelzer, H.: Einseitige Stauungspapille und Störung des Geruchsinnes, Klin. Monatsbl. f. Augenh. **91**:479, 1933.

5. Custodis, E.: Meningeom der Falx und lokalisatorisch irreführendes Foster Kennedy'sches Syndrom. Klin. Monatsbl. f. Augenh. **101**:823, 1938.

6. Cusick, P. L.: Foster Kennedy Syndrome Associated with a Tumor of the Cerebellum, Proc. Staff Meet., Mayo Clin. **13**:433, 1938.

7. Alpers, B. J., and Groff, R. A.: Parasellar Tumors, Arch. Neurol. & Psychiat. **31**:713 (April) 1934.

8. Marchesani, O.: Symptomatologie des Nervus opticus, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 4, p. 58.

9. Kennedy, F.: A Further Note on the Diagnostic Value of Retrobulbar Neuritis in Expanding Lesions of the Frontal Lobes, J. A. M. A. **67**:1361 (Nov. 4) 1916.

10. Schloffer, H.: Erwägungen über die operative Entlastung des intrakraniellen Optikusabschnittes, Med. Klin. **1**:421, 1934.

on the left side were less pronounced. Schloffer concluded that atherosclerosis of the internal carotid arteries must be included in the differential diagnosis of the causation of the Foster Kennedy syndrome. He expressed the opinion that the presence of a nasal field defect should favor a diagnosis of arteriosclerotic disease of the carotid arteries rather than a neoplasm. Glees [11] reported 3 cases in which he believed that the syndrome was secondary to compression of the optic nerves by arteriosclerotic vessels. He offered as an explanation of the pathogenesis of the syndrome in these cases compression of the optic nerve on one side followed by local circulatory impairment in the optic nerve on the other.

11. Glees, M.: Dem Foster Kennedy'schen Syndrom-ähnliche Veränderungen der Sehnerven durch Arteriosklerose, Klin. Monatsbl. f. Augenh. **100**:865, 1938.

SUMMARY

The case reported indicates that arachnoiditis of the optic chiasm and optic nerves is capable of producing the syndrome of atrophy of the optic nerve of one eye and papilledema in the other eye (Foster Kennedy syndrome) and in this respect serves to enlarge the field of possible causes in the production of this syndrome.

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HYPERTENSIVE RETINOPATHY ASSOCIATED WITH ADRENAL MEDULLARY TUMOR (PHEOCHROMOCYTOMA)

A NEW CLINICAL ENTITY

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A review of the available literature reveals that about 35 cases of pheochromocytoma have been reported to date. Although definite ocular changes are associated with the presence of these chromaffin cell tumors, they have received little attention. Many reports failed to mention the results of ophthalmic examination; some, however, described briefly the concomitant pathologic process observed in the eye. To my knowledge, the case of adrenal medullary tumor to be reported is the first in which photographic records of the fundi were made after operation, illustrating the gradual disappearance of retinal changes. When the patient was seen three and one-half years after removal of the tumor, the retinas were normal in appearance.

Chromaffin tumors (pheochromocytoma, chromaffinoma, paraganglioma) arise from the chromaffin tissue of the embryonic sympathetic nervous system (Tassman [1]) and may be located in any of the paraganglions. The adrenal medulla is composed of chromaffin cell tissue, and hyperplasia of this structure probably represents the rarest form of adrenal tumor (Grollman [2]).

The general syndrome produced is characterized by "evidence of instability of the sympathetic nervous system, hypertension or paroxysmal hypertension, glycosuria, periodic attacks of tachycardia, vasoconstriction and vasodilatation of peripheral vessels, headaches, nausea, vomiting, sensations of constriction in the epigastrium with dyspnea, and susceptibility to shock (Labbé, Tinel and Doumer [3]). These authors were first to observe the phenomenon of paroxysmal hypertension associated with pheochromocytoma. They described the typical case as marked by periodic attacks occurring at intervals of a few hours or in response to a stimulus, such as massage of the tumor mass. During the episodes, which usually last from five to fifteen minutes, apprehension, pallor, sweating, mydriasis, hypertension and hyperglycemia are signal features. Biskind, Meyer and Beadner,[4] in an extensive review of 29 collected cases and 1 personal case, observed conclusively that successful removal of the tumor provides relief of symptoms in the majority of cases, particularly when supported by adequate preoperative and postoperative care.

The cells of the pheochromocytoma have been shown to produce epinephrine [2] or an epinephrine-like substance. This was demonstrated for the first time in the circulating blood during a crisis by Hyman and Mencher.[5] According to Tassman,[1] epinephrine secreted by the medulla is a pressor substance and is known to cause vasoconstriction, elevate blood pressure and stimulate the sympathetic nervous system. The medullary chromaffinoma, therefore, by releasing the accumulation of pressor substance into the blood stream, may give rise to attacks of paroxysmal hypertension characteristic of an epinephrine reaction. The quantity of epinephrine contained in these tumors has been measured and provides convincing evidence of the correlation between the two phenomena.

In the eyes, this situation is often manifested by changes typical of general hypertension, as follows: dilated pupils; pallor of the conjunctivas; papilledema; hyperemic disks with indistinct margins; engorged veins, irregular in caliber; thinned, conspicuously tortuous arteries

From the Department of Ophthalmology, Mount Zion Hospital.

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1. Tassman, I. S.: The Eye Manifestations of Internal Diseases, St. Louis, C. V. Mosby Company, 1942, pp. 55 and 451.

2. Grollman, A.: The Adrenals, Baltimore, Williams & Wilkins Company, 1936, pp. 333-336.

3. Labbé, M.; Tinel, J., and Doumer, E.: Crises solaries et hypertension paroxystique en rapport avec une tumeur surrénale, Bull. et mém. Soc. méd. d. hôp. de Paris **46**:982-990 (June 23) 1922.

4. Biskind, G. R.; Meyer, M. A., and Beadner, S. A.: Adrenal Medullary Tumor: Phaeochromocytoma Cured by Surgical Intervention; Clinical Management; Analysis of All Reported Operated Cases, J. Clin. Endocrinol. **1**:113-123 (Feb.) 1941.

5. Hyman, A., and Mencher, W. H.: Pheochromocytoma of Adrenal Gland, J. Urol. **49**:755-776 (June) 1943.

with increased light reflex in the fundus, and compression of the veins where they are crossed by the arteries. Small, flame-shaped hemorrhages and star-shaped figures are not uncommon, and perivasculitis, fibrinous exudate and fine spots of degeneration may appear in the fundi. On the basis of these changes, the ischemia resulting from thinning of the retinal arteries has been suggested by Oppenheimer and Fishberg [6] as the cause of "retinitis albuminurica." Tassman observed occasional exophthalmos. During the height of an attack, Hyman and Mencher found that the retinal arteries were somewhat narrower than normal and that they indented the veins slightly, were rather regular in caliber and showed a bright, irregular reflex. A slight propulsive pulse was noted. In 1 of their 4 cases the fundi were normal except for moderately thinned arteries.

REPORT OF A CASE

Mrs. A. B., aged 31, a saleswoman, entered Mount Zion Hospital on Nov. 11, 1939. She complained of attacks of nausea, vomiting and diaphoresis associated with headaches of seven years' duration. These attacks had become severe four weeks prior to her admission. Headaches consisted of dull, diffuse pains, which lasted for several days and recurred every three or four months. These were accompanied with nausea, vomiting and diaphoresis. The attacks lasted from five to fifteen minutes. A diagnosis of migraine had been made two years previously, and ergotamine tartrate had been administered, with relief, for several months; improvement was followed by renewed episodes of increased severity, which did not respond to this medication. The patient noticed that wearing a tight girdle or stooping forward appeared to incite an attack. She was seen by my associates and me two weeks after a particularly severe episode; at this time the diagnostic impression, based on the results of physical examination (including ophthalmoscopic investigation) and laboratory tests, was that of adrenal medullary tumor. Two years previously a physician had observed a slight thickening of the retinal arteries.

The past history was irrelevant. In the interval prior to operation the only unusual observations made on physical examination were a systolic murmur, an accentuated aortic sound, slight thickening of the peripheral arteries, a tumor mass in the region of the right kidney and changes in the optic fundi. Typical attacks were produced by massaging the tumor in the right flank. These consisted in a sensation of constricting pressure in the lower part of the chest with a pounding headache; a temporary increase in the respiration rate and the pulse rate, followed by a fall to 60 or 70 a minute; a rise in blood pressure to from 260 to 290 systolic and from 150 to 180 diastolic; cold and perspiring hands and feet, and nausea and vomiting. Paroxysms subsided within five minutes; the pulse rate rose to from 90 to 100 a minute; the blood pressure fell to from 100 to 130 systolic and from 80 to 95 diastolic. The hands and feet became warm, and the patient felt exhausted. Laboratory studies showed an essentially normal condition except for hyperglycemia, glycosuria and a dextrose tolerance curve of diabetic type. There was slight tachycardia. Roentgenograms of the skull and chest were normal. Renal function was normal. Retrograde pyelograms revealed depression of the upper calices of the right kidney as if by a tumor mass and angulation of the upper end of the right ureter. Psychiatric examination showed fair stability.

Examination of the Eyes.—Initial examination of the eyes was made on November 19, one month previous to operation. The patient gave a history of having no trouble with her eyes except during the attacks of nausea and vomiting associated with headaches. At such times she experienced pain in the eyes, especially on looking upward. Vision became impaired for near and distant objects. She had the sensation of light coming from all directions, which annoyed her. When the attacks subsided, vision returned to normal, although the eyes felt weak for a few hours afterward.

Vision was 20/20 in each eye. External examination showed that the eyes were normal. The pupils were equal in size and reacted promptly to light and in accommodation.

Ophthalmoscopic Examination.—Right Eye: The media were clear. The optic disk was normal in appearance and vertically oval, with well defined margin. The retinal arteries were tortuous and narrow; the relation of the diameter of the arteries to that of the veins was 1:2. The light streak was well marked, giving the arteries the appearance of being definitely pale, with narrow, red borders. The retinal veins were engorged and showed slight tortuosity. The arteries and veins were solid looking. There was definite compression of the veins where they were crossed by the arteries. Many small hemorrhages were seen throughout the retina. These did not appear to be of recent origin; some seemed to be in the process of absorption; most were flame shaped. There were numerous white patches around the macular region, principally of the soft, fluffy, cotton wool type. These appeared in the form of narrow, elongated patches, between which normal, orange-colored retinal tissue was present. In addition, there were small, round, isolated patches. The picture was suggestive of a beginning star-shaped figure around the macula. There was also a large, opaque, white spot, 1.5 disk diameters from the margin of the disk in the direction of 10 o'clock. There was likewise an area of exudate partially covering the inferior temporal artery about 1.5 disk diameters from the margin of the disk, extending to the upper margin of the inferior temporal vein.

Left Eye: The media were clear. The upper and nasal portions of the margin of the disk were definitely blurred. The disk was normal in color. The retinal arteries showed the same changes as those seen in the right eye, except that tortuosity was more pronounced and the arterioles were narrower. Some of the arteries, especially the branches, were covered in their course by exudate. The retinal veins were similar in appearance to those in the right eye. Changes about the macula were more striking. A large, arrow-shaped opacity was present above the macula. There were many narrow, opaque, white patches pointed toward the macula, below and nasal to it. Irregular white patches were also present in the upper temporal quadrant of the retina. There were many small hemorrhages of various sizes around the macula.

6. Oppenheimer, B. S., and Fishberg, A. M.: Association of Hypertension with Suprarenal Tumors, Arch. Int. Med. **34**:631-644 (Nov.) 1924.

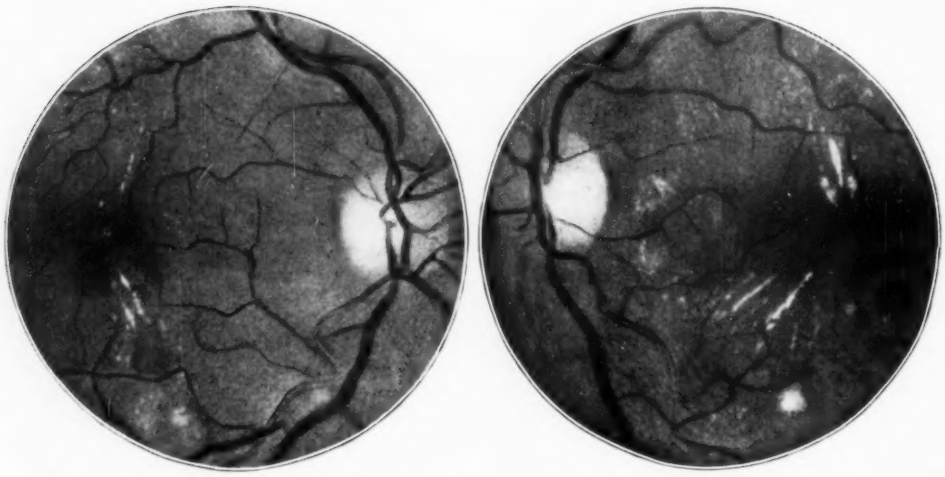

Fig. 1.—Right Eye (on the left): The optic disk is vertically oval with well defined margin. The retinal arteries are tortuous and narrow, the relation of their diameter to that of the veins is 1:2. The light streak is well marked, giving the arteries the appearance of being definitely pale with narrow, red borders. The retinal veins are engorged and show some tortuosity. There is definite compression of the veins where they are crossed by the arteries. Small retinal hemorrhages which were present did not show in the photograph. There are numerous white patches around the macular region, mostly of the soft, fluffy, cotton wool type. These appear in the form of narrow, elongated patches, between which normal, orange-colored retinal tissue is seen. The picture is suggestive of a beginning star-shaped figure around the macula. There is also a large, opaque, white spot 2.5 disk diameters from the disk in the direction of 10 o'clock. An area of exudate partially covers the inferior temporal artery about 1.5 disk diameters from the margin of the disk and extends to the upper margin of the inferior temporal vein.

Left Eye: The upper and nasal portions of the margin of the disk are definitely blurred. The retinal arteries show the same changes as those seen in the right eye, except that tortuosity is more marked and the arterioles are narrower. Some of the arteries, especially the branches, are covered in their course by exudate. The retinal veins have the same appearance as in the right eye. Changes about the macula are more striking. There is a large, arrow-shaped opacity above the macula. There are many narrow, opaque, white patches pointing toward the macula, below and nasal to it. Irregular white patches are also present in the upper temporal quadrant of the retina.

The photographs were taken one month after removal of the tumor. The retinal changes are the same as those present before the operation.

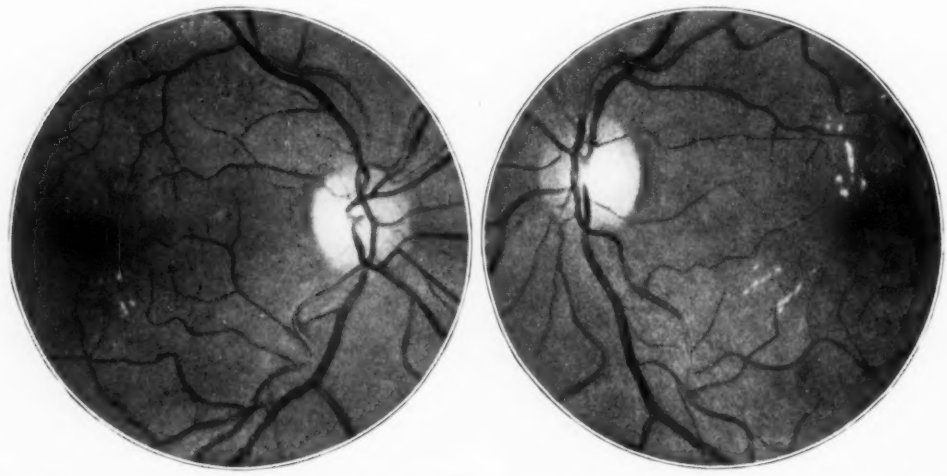

Fig. 2.—Photographs taken two and one-half months later (three and one-half months after the operation), showing definite changes in the fundi: marked diminution of the exudate, especially in the right eye, and a more normal appearance of the retinal vessels, with reduced tortuosity. The arrow-shaped exudate in the left eye, pointing to the upper portion of the macula, is smaller, but still an outstanding feature of the retinal picture. The margin of the left disk has become distinct.

Operation was performed on December 19, one month after our first examination of the eyes. During the preoperative period repeated examinations showed no notable change in the fundi from the original observation.

The details of operation and the preoperative and postoperative care of this patient have been reported elsewhere.[4] The adrenal tumor was successfully removed, and after an extremely critical immediate period of convalescence and return to health the blood pressure was stabilized at 125 to 135 systolic and 85 to 100 diastolic. The dextrose tolerance curve became normal and has remained so.

Histologic sections of the specimen removed at operation revealed true pheochromocytoma, of nonmalignant character. Analysis of venous blood showed approximately 0.08 mg. of epinephrine per cubic centimeter. Epinephrine was not identified on quantitative examination of the tumor tissue and cystic fluid; however, the large amounts of a catechol-like substance recovered may be considered a precursor of epinephrine. Significant is the production by the tumor extract of marked mydriasis of the cat's eye.

One month after operation photographs of the fundi were taken (fig. 1). The changes observed two months previously were still present except for possible reduction in number and size of the retinal hemorrhages.

Photographs taken two and one-half months later (fig. 2) showed definite changes in the fundi: marked diminution of exudate, especially in the right eye, and a more normal appearance of the retinal vessels, with reduced tortuosity. In the left eye the arrow-shaped exudate pointing to the upper portion of the macula was smaller, but still an outstanding feature of the pathologic process in the retina. The margin of the left disk had become distinct.

Photographs taken three months later (fig. 3) showed further improvement in the retinas. Only a few white patches remained. The veins appeared normal, but the arteries were still narrow; the relation of their diameter to that of the veins was 1:2. The arteries remained tortuous and their white markings distinct. Considerable compression of the veins persisted where they were crossed by arteries. The arrow-shaped area of exudate in the left eye pointing to the macula was still present.

One year later ophthalmoscopic examination revealed a normal retina in the right eye. The retinal veins still showed some engorgement, whereas the arteries were normal; no exudate or hemorrhages were present. The left eye revealed similar improvement except for two white vertical stripes where the arrow had been.

Examination of the eyes three and one-half years after removal of the tumor revealed that the retinas were normal in appearance.

COMMENT

Extensive changes in the retinas, characterized by changes in the vessels, hemorrhages and formation of exudate, were observed in a patient with an adrenal medullary tumor (pheochromocytoma). After removal of the tumor the ocular phenomena disappeared.

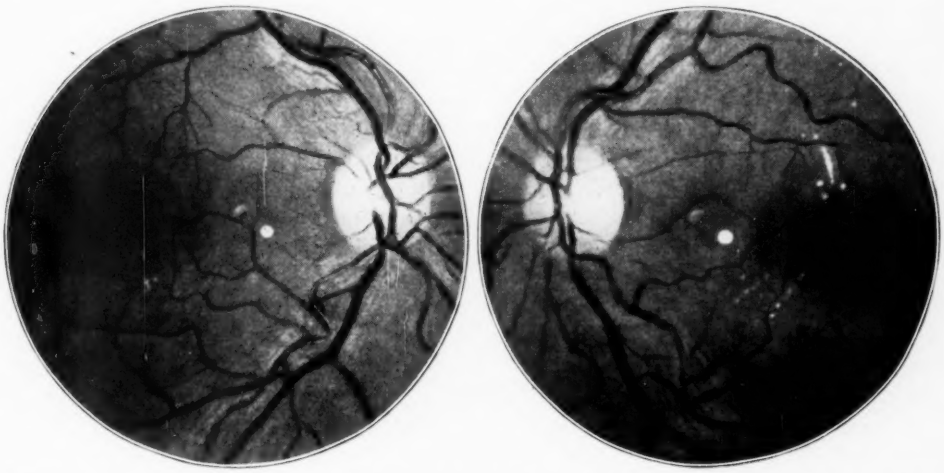

Fig. 3.—Photographs taken three months later (six and one-half months after the operation), showing further improvement in the retinas. Only a few white patches remain. The veins appear normal, but the arteries are still narrow; the relation of their diameter to that of the veins is 1:2. The arteries are still tortuous, with the white markings distinct. Considerable compression of veins persists where they are crossed by the arteries. The arrow-shaped opacity in the left eye pointing to the macula is still present.

There is no definite explanation of the mechanism by which the retinal changes associated with pheochromocytoma are produced. Tassman stated that three principal explanations are given for the ocular manifestations accompanying glandular dysfunction; all have to do in some way with gradual reduction of blood supply and impairment of nutrition. First, the retinal lesions are of vascular origin and are due primarily to changes in the walls of the vessels. Second, they are due to the toxic chemical action on the retinal tissue and vessels of certain products of degeneration which are formed as the result of disease in

another organ—in the present case, the adrenal tumor. Third, a disturbance of metabolism, with breaking down of cellular elements and production of toxins, takes place simultaneously in a primary organ.

Moon,[7] in describing experimental epinephrine shock, stated that large quantities of the injected drug may produce such marked, prolonged arterial constriction that the tissues are deprived of oxygen. Like anoxia of other origin, this type may be expected to be followed by capillary and arteriolar atony and increased endothelial permeability. When permeability is greatly increased, whole plasma escapes; when it is only moderately increased, the smaller protein molecules (albumin) pass rapidly through the relaxed membrane and the larger ones (globulin) tend to be retained. This could account for the retinal hemorrhages and exudates observed and photographed in this study.

In my case, in addition to the adrenal tumor and the paroxysms of hypertension, the only definite observations were the changes in the retinas. It is fair to assume that these changes were produced by the liberation of increased amounts of epinephrine due to the higher activity of the epinephrine-secreting portion of the adrenal gland consistent with tumor growth.

Epinephrine under ordinary conditions exerts the same physiologic effect as sympathetic, not parasympathetic, stimulation. It acts neither on the smooth muscle fibers nor on the sympathetic nerves alone, but directly on the myoneural junction. This means that the splanchnic head is constricted by the drug and that the retinal vessels are likewise constricted. The combined effect would be hypertension and ischemia with malnutrition.

In all cases of pheochromocytoma in which an ophthalmic examination was made changes in the eyes, varying from thinning of the arteries to the extensive pathologic process described in the present case, were noted.

How can this ocular condition be classified? According to Duke-Elder,[8] vascular retinopathies are conditions of the eye in which to the factor of vascular sclerosis, hypertension and toxemia are added. More dramatic and generally distributed alterations ensue in the retinal tissues themselves, characterized essentially by hemorrhages and exudates. All these conditions are dependent on and associated only with hypertension. Without it they do not occur, even in the presence of arteriosclerosis or widespread renal destruction, as when the kidney is almost entirely destroyed by tuberculosis, sepsis or neoplasm.

From both the topographic and the prognostic point of view, Duke-Elder classifies them into the following types: (1) arteriosclerotic retinopathy, (2) renal retinopathy, (3) toxemic retinopathy of pregnancy, (4) malignant hypertensive retinopathy and (5) diabetic retinopathy.

The ocular lesions in my case most closely resembled malignant hypertensive retinopathy. However, there are important differences. The malignant hypertensive type is characterized by extreme papillary and peripapillary edema. In my case there was only blurring of the margin of the left disk and no edema. In none of the cases reported was there marked papillary edema, as is seen in the malignant type. The characteristic clinical picture of intense arterial constriction, hyperemia and massive edema of the disk, with profuse superficial hemorrhages and soft, cotton wool exudates, was lacking. In the present case small hemorrhages and limited formation of exudate were present. The star-shaped figure which is often pronounced in malignant disease was not well developed in this case. In cases of malignant type the picture is that of acute and perilous toxemia; in my case it was one of a slowly progressing condition. The question arises whether if the adrenal tumor were not removed a retinal picture characteristic of malignant hypertension would eventually develop. The answer, of course, is lacking, as no case of adrenal tumor has been reported in which the ocular condition found in the malignant type was present.

The outstanding feature in this case was the disappearance of the pathologic condition of the retina after removal of the tumor. As a rule the picture of malignant hypertensive retinopathy is considered to be, as Duke-Elder expressed it, a "death warrant." There are cases observed over a long period in which renal failure did not develop and partial or complete recession took place.[9] These cases, however, are the exception.

7. Moon, V. H.: Shock: Its Dynamics, Occurrence and Management, Philadelphia, Lea & Febiger, 1942, p. 48.

8. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1940, vol. 3, pp. 2706-2729.

9. Schieck, F.: Netzhautveränderungen bei Nierenleiden und bei Blutdrucksteigerung, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11, pt. 1, pp. 619-630. Floyd, R.: Retinal Changes in Hypertension and in Renal Disease, Arch. Ophth. **6**: 433-444 (Sept.) 1931. Fishberg, A. M., and Oppenheimer, B. S.: Differentiation and Significance of Certain Ophthalmoscopic Pictures in Hypertensive Diseases, Arch. Int. Med. **46**:901-920 (Dec.) 1930.

It is my opinion, therefore, that the case reported presents a retinal entity characterized by the changes in the retina, hypertensive paroxysms and the presence of a pheochromocytoma, in which the pathologic process in the retina disappeared after removal of the tumor. Such an ocular condition should be classified as a hypertensive retinopathy associated with adrenal medullary tumor.

In cases of hypertensive retinopathy with paroxysmal attacks the possibility of a coexisting adrenal tumor should always be considered.

SUMMARY

Pheochromocytoma is a rare form of chromaffin tumor, which is characterized by a train of hypertensive symptoms, including definite ocular changes.

Epinephrine or an epinephrine-like pressor substance is contained in the cells of this tumor. Large amounts of this substance freed into the blood stream cause vasoconstriction, elevate blood pressure and stimulate the sympathetic nervous system, producing paroxysms of hypertension. In the eye, changes are typical of hypertensive retinopathy.

Extensive changes in the retinas, characterized by changes in the retinal vessels, hemorrhages and formation of exudate, were observed in a patient with an adrenal medullary tumor (pheochromocytoma). After removal of the tumor the ocular phenomena disappeared, as illustrated by the serial photographs. There is no definite explanation of the mechanism by which the retinal changes are produced.

I feel that the ocular condition in this case is a new clinical entity and should be classified as hypertensive retinopathy associated with an adrenal tumor.

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CHANGES OF LABYRINTHINE EXCITABILITY IN LESIONS OF OPTIC TRACT OR EXTERNAL GENICULATE BODY

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Little is known as to whether retinal impulses are able to influence the excitability of the vestibulo-ocular reflex arc. Ohm [1] observed that postrotational nystagmus is different in the dark and in the light. It is, however, not clear from his experiments whether the differences observed were due to the changes in the illumination or to the influence of fixation. In experimental studies of Spiegel,[2] it was shown that the effect of stimulation of the occipital lobe on ocular movements is changed by a lesion of the vestibular nuclei. While horizontal conjugate deviation of the eyeballs predominated as long as the vestibular nuclei were intact, the stimulation of the occipital lobes following bilateral lesions of the vestibular nuclei produced chiefly vertical ocular movements. These experiments were interpreted as indicating that the impulses for horizontal ocular movements descending from the occipital lobe reach the nuclei of the ocular muscles after passing through a relay station in the vestibular nuclei. This corticofugal system from the occipital lobe to the vestibular nuclei could serve for conduction of impulses originating in the occipital lobe and influencing the vestibulo-ocular reflex arc. The existence of such an influence was demonstrated in further experiments by one of us with Dr. Wycis,[3] in which unilateral destruction of the area striata was followed by differences in the postrotatory nystagmus on clockwise and on counterclockwise rotation, the postrotatory nystagmus to the side of the lesion lasting much longer than that directed to the opposite side. This phenomenon seems related to the directional preponderance observed by Fitzgerald and Hallpike [4] on caloric stimulation in cases of brain tumor. It suggested that elimination of the centripetal impulses entering the area striata may have a similar effect. In order to test this possibility, experiments were performed in which the effect of unilateral lesions of the optic tract or of the external geniculate body was studied.

METHOD

Lesions of the optic tract or of the external geniculate body were produced electrolytically in cats under pentobarbital sodium (nembutal) anesthesia, the needle being introduced by means of a Horsley-Clarke stereotaxic apparatus; or the optic tract was cut after it had been exposed by the technic of the overhanging brain developed by Karplus and Kreidl.[5] Postrotatory nystagmus was produced on an electrically driven rotating chair that insured uniform stimulation by 10 rotations in twenty seconds. The duration of this nystagmus and the number of jerks were determined, and their amplitude was estimated. The animals were subjected alternately to 10 clockwise and to 10 counterclockwise rotations, each series of rotations being performed three times. The cats were first tested for several days to ascertain fluctuations in the excitability, animals with marked differences on clockwise and on counterclockwise rotation being discarded. The examination was repeated on the day following the operation and subsequently at intervals of from two to three days in the first one or two weeks and later at weekly or longer intervals for periods up to four months.

RESULTS

In principle the same changes were observed, whether the lesions were placed in the optic tract or in the external geniculate body. Similarly as in the striatal lesions, a marked difference between the postrotatory nystagmus following clockwise rotation and that following counterclockwise rotation was observed postoperatively. There was a "directional preponderance," the nystagmus directed with its fast component toward the side of the operation being distinctly more intense than that directed to the opposite side. The duration of the postrotatory nystagmus to the side operated on reached up

From the Department of Experimental Neurology, Temple University Medical School, Philadelphia.

1. Ohm, J.: Zur Augenzitterkunde, Arch. f. Ophth. **125**:245, 462 and 554, 1930; **126**:221, 1931.

2. Spiegel, E. A.: Role of Vestibular Nuclei in the Cortical Innervation of the Eye Muscles, Arch. Neurol. & Psychiat. **29**:1084 (May) 1933.

3. Wycis, H. T., and Spiegel, E. A.: Influence of Cortical Areas upon the Vestibulo-Ocular Reflex Arc, to be published.

4. Fitzgerald, G., and Hallpike, C. S.: Observations on the Directional Preponderance of Caloric Nystagmus Resulting from Cerebral Lesions, Brain **65**:115, 1942.

5. Karplus, J. P., and Kreidl, A.: Gehirn und Sympathicus, Arch. f. d. ges. Physiol. **129**:138, 1909.

to three times, the number of its jerks up to four times, the corresponding values of the nystagmus to the normal side; often also the amplitude of the jerks was larger when the postrotatory nystagmus to the side operated on was elicited than when the oppositely directed nystagmus was produced. On comparison with the preoperative values one found either increase of the duration and number of jerks of the nystagmus to the side operated on (see cat 128) or decrease of the nystagmus to the normal side (see cat 124) or a combination of these two phenomena. Occasionally there was an increase of the reaction to both sides, but also in such instances there was still the preponderance to the side operated on (see cat 135). The changes were often demonstrable as soon as twenty-four hours after operation, as illustrated by the following experiment.

Cat 132	Postrotatory Nystagmus to Left (after 10 ↷)		Postrotatory Nystagmus to Right (after 10 ↶)	
	Duration	No. of Jerks	Duration	No. of Jerks
12/7/44	14-15 sec.	15	12-14	14-15
12/7/44	Severance of left optic tract on overhanging brain			
12/8/44	27-30	32-41	10-13	6-12
	Amplitude of nystagmus to left definitely larger than that of nystagmus to right.			
	Anatomic examination revealed complete severance of the left optic tract close to the chiasm.			

In all tabulations ↷ means clockwise rotations and ↶ means counterclockwise rotations.

In some cases the excitability of the vestibulo-ocular reflex arc was lowered on the first postoperative day and the phenomena of increased excitability described appeared one to two days later. This may be due partly to prolonged action of the anesthetic, partly to "shock." In this stage, some cats also showed an increased latent period of the reaction (see cat 128), indicating that increase of the latent period on testing the labyrinth may be caused by lesions of the central nervous system, while some clinicians assume that the increase of the latent period on caloric examination of the labyrinth indicates hypoexcitability of the receptor organ (Brunner [6]).

The duration of the "directional preponderance" chiefly depends on the extent of the lesion. While in more or less complete interruption of the centripetal impulses from the homolateral halves of the retinas a prevalence of the nystagmus to the side operated on could be observed for several months, a return to the normal values took place within a week in incomplete lesions.

6. Brunner, H.: Allegemeine Symptomatologie der Erkrankungen des Nervus vestibularis, in Alexander, G., and Marburg, O.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1924, vol. 1, p. 939.

If one tests the labyrinthine excitability repeatedly with test-free intervals of a few days only, the influence of habituation is added to that of the operative procedure; it tends to lower the labyrinthine excitability bilaterally. If the examinations on the rotating chair are interrupted for a longer time, the excitability of both labyrinths again rises (see end of protocol of cat 128).

The described effect of elimination of retinal impulses may be illustrated by the following abbreviated protocols.

Cat 124	Postrotatory Nystagmus to Left Following 10 Rotations ↷		Postrotatory Nystagmus to Right Following 10 Rotations ↶		
	Duration	No. of Jerks	Duration	No. of Jerks	
4/24/44	14 sec.	21-25*	13-15	18-20	
4/27/44	Electrolytic lesion of right optic tract				
4/28/44	Questionable reaction		20-23	2-10 (undulations with small amplitude)	
4/29/44	6-10	4-11	4-18	15-21	
5/ 1/44	5-9	8-10	12-14	17	
5/ 2/44	11-12	13-17	15-17	16-18	
5/ 8/44	9-11	4-8	12-13	5-14	(fatigue after several tests)
5/10/44	9-13	9-13	10-12	12-14	
5/15/44	9-14	11-13	10-13	5-12	(fatigue after several tests)
5/22/44	11	7-8	10-11	9-10	
5/31/44	10	8	10-11	8-9	(labyrinth reflex easily fatigued)

* In this as well as in the other protocols, the figures represent maximum and minimum values in three tests.

Histologic examination revealed an oval area of demyelination in the dorsolateral part of the optic tract; however, the lesion is incomplete, leaving part of the fibers of the optic tract intact.

No. 128	Postrotatory Nystagmus to Left Following 10 Rotations ↷		Postrotatory Nystagmus to Right Following 10 Rotations ↶		
	Duration	No. of Jerks	Duration	No. of Jerks	Comment
6/ 7/44	12 sec.	15-18	11½-12	19-22	
6/ 8/44	Puncture of left cerebrum without lesion of external geniculate ganglion				
6/ 9/44	7-13 (fatigue on repeated stimulation)	10-21	10-14	17-20	
6/12/44	5-7	9-10	6-9	10-14	
6/14/44	5-9	6-9	5-6	7-10	
6/16/44	5-6	4-6	6-8	8-11	
6/21/44	4-5	4-5	4-5	6	
6/22/44	Electrolysis of right external geniculate body				
6/23/44	7-10 (latent period 5 sec.)	4-6 (slow undulations)	0-13 (latent period 5 sec.)	0-4 (very small amplitude)	Chemosis of lids
6/26/44	5-8	3-5	11-16	15-22	Nystagmus immediately following rotation
6/28/44	5-6	5-6	12	14-15	Large amplitude in both directions, amplitude to left less than to right
6/30/44	3-4	2-3	10-13	11-17	
7/ 5/44	4-5	2-3	15	10-11	
7/10/44	4	4	11	12	
7/14/44	0-5	0-5	7-10	6-7	
7/19/44	4	5	7-8	6-12	
7/24/44	4-5	3-5	6-7	8-9	
8/ 7/44	3	2	8-9	9	
8/24/44	2-3	1-2	8-10	8-9	
10/26/44	7-10	8-12	10-12	12-13	

Histologic examination revealed nearly complete destruction of the right external geniculate body (fig. 1).

Cat 135	Nystagmus to Right (after 10 rotations ↶)		Nystagmus to Left (after 10 rotations ↷)	
	Duration	No. of Jerks	Duration	No. of Jerks
12/28/44	7-7.5	10	7.5-9	10-12
12/29/44	5-8	5-7	5-7	3-5
1/ 2/45	5	4-6	5-7	4-5
	Electrolytic lesion of right external geniculate ganglion			
1/ 3/45	20	38-40	12-14	22-26
1/ 5/45	22-25	45-46	7-9	12-16
1/ 8/45	20-24	32-39	11-13.5	21-29
1/11/45	18-22 (large amplitude)	27-30	10-12 (small amplitude)	11-23

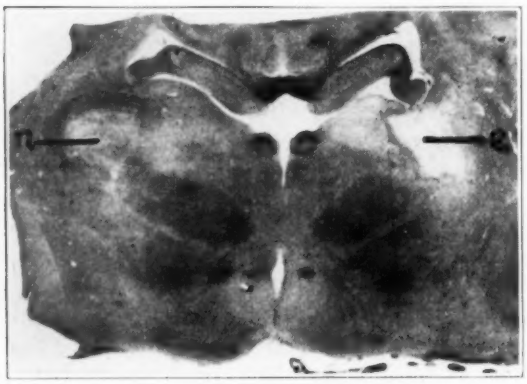

Fig. 1 (cat 128).—Electrolytic lesion (*e*) of right external geniculate ganglion; normal left external geniculate ganglion, *n*.

In control experiments punctures were performed at the same coordinates as in the electrolysis experiments, but without the electrolytic lesion. These controls showed that punctures through the parietal lobe penetrating the internal capsule (or cerebral peduncle) so that the optic tract was reached or through the white matter of the hemisphere above the external geniculate ganglion produced no directional preponderance or at the most a slight difference of a few days' duration, but never the marked long-lasting differences observed in complete or extensive lesions of the optic tract or external geniculate body.

Cat 136	Nystagmus to Right (after 10 rotations ↶)		Nystagmus to Left (after 10 rotations ↷)	
	Duration	No. of Jerks	Duration	No. of Jerks
1/ 8/45	12-14 sec.	25-33	12-13	23-26
1/ 9/45	12.5-13	19-28	11	18-20
1/10/45	Puncture of right cerebrum, reaching right external geniculate body, without electrolysis (fig. 2)			
1/11/45	13	17	7-10	8-16
	Markedly reduced reaction on repetition of test			
1/12/45	10-12	16-24	10-11	14-20
1/15/45	9-10	12-18	9-13	20-21

The postrotatory nystagmus following clockwise rotations chiefly depends on stimulation of the left labyrinth and its reflex arc to the eye muscles, the nystagmus following counterclockwise rotations on stimulation of the corresponding structures on the right side. The preponderance of the nystagmus toward the side of operation in our experiments obviously is not due to changes of excitability of the peripheral receptor, since reflex impulses from the retinas cannot act directly on the peripheral labyrinth. They must act on parts of the vestibulo-ocular reflex arc, either on the vestibular nuclei or on the nuclei of the ocular muscles or the intercalated nuclei of the reticulate substance. This problem of the site of this interference will be approached in further studies. At present it may, however, be stated that retinal impulses seem to have a certain tonic influence on the vestibulo-ocular reflex arc. Our experiments showed that lesions of an optic tract or external geniculate body increase the postrotatory nystagmus to the same side; sometimes they also depress the nystagmus to the opposite side. These observations seem to indicate that impulses from the homolateral halves of the retinas have an inhibitory effect on vestibulo-ocular reflexes tending to produce nystagmus to the same side, while a facilitating effect on labyrinthine reflexes acting in the opposite direction is less regularly demonstrable.

SUMMARY

Lesions of the optic tract or of the external geniculate body were produced electrolytically in cats under anesthesia with pentobarbital sodium, the needle being introduced by means of a Horsley-Clarke stereotaxic apparatus; or the optic tract was cut after it had been exposed by the technic of the overhanging brain. A marked difference between the postrotatory nystagmus following clockwise rotation and that following counterclockwise rotation was observed postoperatively. The duration of the postrotatory nystagmus to the side operated on reached up to three times, the number of its jerks up to four times, the corresponding values of the nystagmus to the normal side. This prevalence of the nystagmus to the side operated on could be observed up to several months following the operation. These observations seem to indicate that impulses from the homolateral halves of the retinas have an inhibitory effect on vestibulo-ocular reflexes tending to produce nystagmus to the same side.

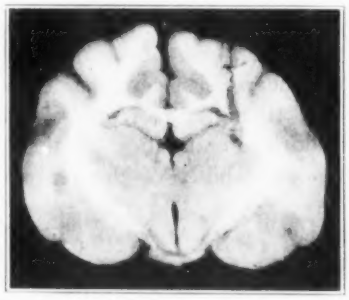

Fig. 2 (cat 136).—Control experiment. Puncture of right cerebral hemisphere without electrolytic lesion of external geniculate ganglion.

OCULAR VARICELLA

REPORT OF A CASE OF CORNEAL PHLYCTENULE

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The ocular complications of varicella (chickenpox) are rare indeed. This impression is supported by the sparsity of the ophthalmic literature dealing with the disease. Conjunctival vesicles [1] exhibiting phlyctenular-like pustules [2] have occasionally been reported to invade the cornea. These may develop into ulcer excavations [3] with swollen, reddish brown margins. Minimal edema of the lids [4] is also frequently demonstrable.

Corneal complications are more infrequent. A direct corneal lesion is most unusual but may appear relatively late in the course of the disease. Oppenheimer, in 1905,[5] reported a case of a vesicular lesion which developed into a shallow ulcer. Pickard,[6] in 1936, reported a case of interstitial infiltration with associated endothelial changes. Rosenbaum,[7] in 1943, described a similar case in which was exhibited a shallow central corneal ulcer with minor infiltration of Descemet's membrane and the endothelium. Superficial keratitis has been associated with chickenpox.

In a review of the information available concerning the ocular complications of varicella several facts seem worthy of summary: (1) The ocular sequelae appear late in the course of the disease; (2) the area involved is small and circumscribed, (3) associated iridocyclitis is absent, and (4) recovery is rapid and complete. The last factor serves to differentiate the ocular changes of varicella from those of smallpox.

REPORT OF CASE

A 30 year old resident physician at the University Hospital was first examined by us on March 10, 1942. The patient's chief complaints were diminution of visual acuity in the left eye, severe photophobia and pain.

Past History.—The past history was not contributory to the present illness.

Present Illness.—The physician began to experience severe general malaise, headache and discomfort in the extremities and joints approximately sixteen to seventeen days after his son had been in contact with a patient with a moderately severe attack of chickenpox. Within twenty-four to thirty-six hours the physician's chest and trunk were involved in a rash suggestive of varicella. This was so pronounced that it closely resembled smallpox except for its polymorphous character. The temperature was 102 F. the first day and 103 F. the second and third days and then slowly fell, but the fever persisted much longer than usual. The lesions were excessive in number. The patient was most uncomfortable, with discomfort of the extremities and head. A minimal, but definite, degree of meningismus was demonstrable the first three or four days of the illness. On approximately the seventh day of the disease the physician noted a moderate degree of photophobia in the left eye on awakening. This became intense and uncomfortable, rapidly changing to a foreign body sensation. A minimal amount of thick mucoid discharge was experienced. The ophthalmologist was called to the home on the eighth day, at which time the results of ophthalmic examination were recorded and an artist was secured at once to reproduce the appearance of the ocular lesion.

Ophthalmologic Examination.—Visual acuity was 6/6 in the right eye and 6/12 + 2 in the left eye. Scattered vesicular lesions were present on the face, lids and scalp. The lesions on the lids presented several stages of development, but the majority manifested purulent change. A slight amount of edema of the lids was noted in both eyes, but the swelling was more severe in the left eye. The eyes were straight in the primary position. The cover test disclosed minimal exophoria. The direct, consensual and accommodative reflexes were active and equal in the two eyes. The conjunctiva of each eye revealed several small vesicular areas, each of which possessed a small areola of hyperemia. Slight chemosis was present in both eyes. A faint pericorneal flush was noted in the left eye, being more conspicuous in the 6 o'clock meridian. There was suggestive corneal anesthesia of the left eye. The cornea of the right eye was normal except for a few scattered pinpoint areas which stained with 2 per cent fluorescein. A binocular loupe was necessary to visualize these areas. They were also present in the left eye, but, in addition, there was a definite zone of infiltration 1 to 0.5 mm. in diameter and 1 to 2 mm. from the limbus in the 6 o'clock meridian. This area was raised and stained diffusely with fluorescein. Examination with the slit lamp revealed minimal loss of superficial epithelium over the area of infiltration, which spread diffusely into the superficial stroma for about 2.5 mm. The cornea was thickened superficially, but no deep lesions could be demonstrated.

1. Hilbert, R.: Centralbl. f. prakt. Augenh. **26**:39-41, 1902.
2. Yan, C.: Klin. Monatsbl. f. Augenh. **74**:484-488, 1925.
3. Sommer, G.: Wchnschr. f. Therap. Hyg. d. Auges **12**:205-206, 1909.
4. Pearson, A. C.: Brit. M. J. **1**:1492, 1903.
5. Oppenheimer, E. H.: Deutsche med. Wchnschr. **31**:833, 1905.
6. Pickard, R.: Brit. J. Ophth. **20**:15-18, 1936.
7. Rosenbaum, H. D.: Am. J. Ophth. **26**:53-56, 1943.

No aqueous flare was noted in the left eye. The iris was normal in this eye. The fundus was normal in both eyes except for slight hyperemia of the disk and retina.

Therapy was supportive, consisting in the instillation of an aqueous solution of metaphen (1:2,500), 1 drop in each eye four times a day, and ice compresses to the left eye four times a day.

Under daily observation, the zone of infiltration surrounding the phlyctenular-like lesion receded rather promptly. The ulcerated surface, a small but elevated crater, assumed a brownish red color. This seemed to be the result of accumulation of cellular debris. The phlyctenule disappeared within a period of six to seven days, but a minor degree of corneal anesthesia persisted for at least ten days. There was complete recovery of visual acuity in the left eye, as well as absence of corneal scarring, one month after the onset of the ocular lesion.

COMMENT

In the light of the associated corneal anesthesia, the possibility of herpetic lesions of the cornea cannot be ruled out. The occurrence of such a complication is not completely dismissed in the literature and cannot be disproved in this case. The associated conjunctival vesicles and cutaneous lesions seem to support the diagnosis of varicella. The literature indicates a frequent association between herpes and varicella.

SUMMARY

Several factors revealed in the present case support similar observations mentioned in the literature: (1) the relatively late onset of ocular sequelae, (2) the small area involved, (3) the rather circumscribed character of the lesions, (4) the absence of iridocyclitis and (5) the completeness and rapidity of healing.

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Ocular lesion associated with varicella.

GONIOSCOPY SIMPLIFIED BY A CONTACT PRISM

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Examination of the angle of the anterior chamber of the human eye is shortened and simplified by the use of a contact prism which does not require unusual illuminating and microscopic equipment. The prism, which does not extend beyond the limbus, is placed directly on the cornea and is held in apposition with the cornea by pressure exerted through a flexible spring mounted on a speculum. A capillary film creates optical continuity between prism and cornea and, at the same time, lubricates the contact surfaces. Contacting surface *A* (fig. 1)

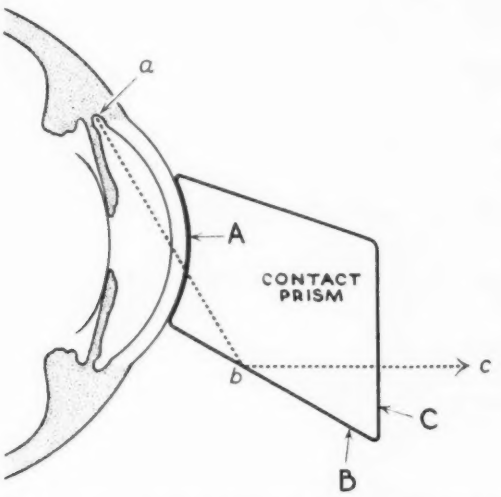

Fig. 1.—Cross section showing prism gonioscope on the cornea, with ray of light emerging from the angle of the anterior chamber.

conforms to the curvature of the anterior surface of the average cornea. Surfaces *B* and *C* form an internal reflecting prism. A ray of light from *a* in the angle of the anterior chamber is reflected at *b* to the observer, at *c*.

The prism is pivoted and suspended by fine wire springs, *a* (fig. 2 *A* and *B*), from the carriage, *b*, which rotates within a groove at the wide end of a hollow truncated cone, *c*. Extra thickness on the under side of the cone at *d* lifts the speculum above the lower lid to counteract the normal projection of the upper lid over the cornea. The flange, *e*, conforms to the curvature of the sclera and retains the instrument by fitting under the lids. The handle, *f*, is used for insertion of the speculum, stabilizing it when the prism is rotated, and for manipulation to help in changing the angle of view into the chamber angle.[1]

Both prism and speculum are made of transparent plastics and, therefore, are relatively unbreakable.[2]

Two drops of a 0.5 per cent solution of tetracaine hydrochloride is administered to the eye which is to be examined. The patient is seated at the slit lamp biomicroscope, as for the usual examination with the slit lamp. A drop of methyl cellulose solution [3] is placed on the contacting surface of the prism. The speculum, with the handle below, is introduced under the upper lid while the patient looks down. Then, as he looks straight ahead, the lower lid is slipped over the flange of the speculum. The patient's head may be tilted backward to assist in lifting the upper lid and to rotate the eye to the center of the prism. The binoculars are directed and focused toward the mirror image of the chamber angle, which appears to be situated behind the reflecting surface of the prism.

The chamber angle is illuminated by the slit lamp beam. When the reflecting surface is above or below the pupil, the light beam must be directed to the desired area by the reflecting surface (fig. 3 *A*). To accomplish this, the slit lamp is placed as nearly coaxial with the biomicroscope as possible. When the reflecting

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1. A similar prism mounted on a flexible handle, and with an accessory magnifying surface for use with the loupe or the unaided eye, has been described. This earlier development is used similarly but is held in place by hand (Allen, L.: A New Contact Lens for Viewing the Angle of the Anterior Chamber of the Eye, Science **99**:186 [March 3] 1944.

2. Bausch and Lomb Optical Company, Rochester, N. Y., will make the prism gonioscope available.

3. The preparation used was Methocel (Dow Chemical Company). One per cent of the 4,000 centipoise type of methyl cellulose in a solution of sodium chloride, recently introduced for use with contact lenses by Kenneth C. Swan, M.D., is water soluble, nontoxic, highly transparent and lubricating. If this solution is not available, isotonic solution of sodium chloride may be substituted.

surface is to either side of the pupil, the best illumination is achieved by refracting the light beam directly through the anterior surface of the prism (fig. 3 *B*). In both these positions the slit lamp beam is kept on the same horizontal plane with the objective lenses of the biomicroscope. Transillumination through the sclera and limbus is also practical.

The angle of view is changed, in order to see behind the iris hump or to view more of the posterior surface of the cornea, by having the patient shift his eye toward or away from the reflecting surface of the prism. This change of angle may be increased by adjusting the handle of the instrument. More than one fourth of the circumference of the chamber angle is visible with the prism in any one position. To examine other portions, the prism is rotated on the cornea.

With experience, it will be found that the entire examination time will correspond roughly to that needed for other examinations with the slit lamp.

A few cases have been encountered in which the gonioscope did not rest close enough to the globe. In such instances the examiner supports the instrument by the handle with one hand while adjusting the biomicroscope with the other. This additional manipulation does not make the examination difficult or notably prolong it.

Any negative pressure (present in the use of other types of gonioscopes) between the prism and the eye is avoided by the flexible linkage of the prism to its support. There is, therefore, no distortion in the appearance of the chamber angle and no disturbance of the blood supply to the anterior segment of the eye. Optical continuity between cornea and prism is maintained easily because of the capillary film and flexible mounting. If this continuity is temporarily interrupted, it can be reestablished immediately by shifting of the patient's eye.

None of the eyes examined has shown abrasion of the corneal epithelium resulting from direct contact of this prism with the cornea. The prism has been left on the normal eye for as long as twenty minutes without disturbance of the tissues and without apparent discomfort to the patient.

University Hospitals.

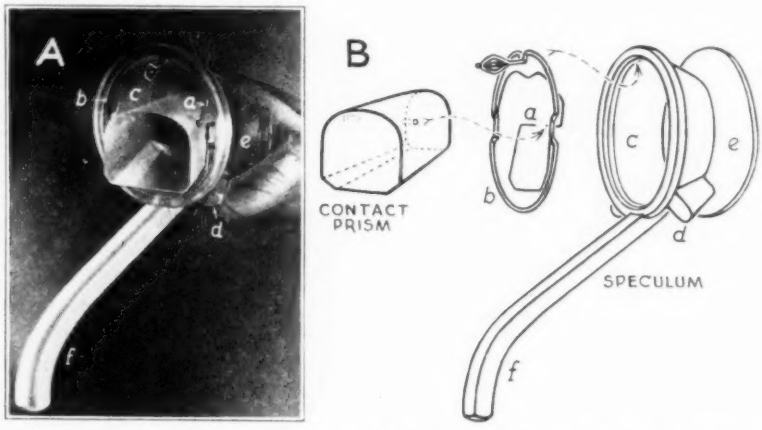

Fig. 2.—*A*, photograph of the prism gonioscope in place on the eye; *B*, component parts of the prism gonioscope.

Fig. 3.—Illumination of the anterior chamber during examination with the prism gonioscope (*A*) by reflection within the prism, while the examiner is viewing the upper or lower portion, and (*B*) by refraction, while the examiner is viewing the lateral portions.

DEVELOPMENT OF SECRETORY FUNCTION OF CILIARY BODY IN THE RABBIT EYE

EVALUATED FROM ASCORBIC ACID CONCENTRATIONS AND CHANGES IN VOLUME

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BLANCHE JACKSON, PH.D.
AND
T. L. TERRY, M.D.
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Recent studies on the adult rabbit have indicated that electrolytes enter the anterior chamber of the adult rabbit eye as a result of a secretory process.[1] In order to evaluate one of the various possible etiologic factors responsible for the characteristic ocular maldevelopment in approximately 12 per cent of infants born extremely prematurely,[2] it seemed important to determine when the intraocular fluid assumes its adult character of a secretion.

Since the high concentration of ascorbic acid in the aqueous humor of adult animals is thought to be maintained by secretion,[3] one approach to the solution of the problem has been to measure the time at which ascorbic acid reaches the concentration characteristic of the adult intraocular fluid. To obtain these data, ascorbic acid concentrations in the aqueous humor were determined successively at intervals after birth and the results compared with changes in volume of the anterior chamber of the whole eye.

EXPERIMENTAL STUDY

Materials and Methods.—The young rabbits used in these experiments were either born in the laboratory farm or obtained within a few days after birth from a local animal dealer. They were housed with the mother until weaning. The diet of the mother consisted of Purina rabbit pellets, fresh vegetables and hay. Both food and water were allowed as desired.

Samples of aqueous humor were obtained with local anesthesia (tetracaine hydrochloride) by means of calibrated micropipets. These pipets were made of capillary Pyrex tubing and the shank of a no. 27 hypodermic needle which was cemented into one end. On insertion of the needle through the cornea into the anterior chamber, the aqueous humor flowed into the pipet through capillary action. The volume of the aqueous humor withdrawn by means of this technic was estimated to represent over 90 per cent of that present. In the younger animals it was necessary to make a short incision in the skin in the region of the palpebral fissure in order to separate the lids before withdrawing the aqueous humor. For animals less than 2 weeks of age, several samples of aqueous humor were pooled. Samples of blood were obtained by cardiac puncture. The Mindlin-Butler micromethod[4] for the determination of ascorbic acid (reduced) was used for both blood and aqueous humor. For determinations of the ascorbic acid in the aqueous humor, samples of 0.010 to 0.085 cc. were employed; for the determinations of the vitamin in the blood 0.1 to 0.2 cc. was used. In all instances the samples of aqueous humor were placed in 5 per cent metaphosphoric acid immediately after collection. The Cenco-Sheard-Sanford photelometer was used for the colorimetric determinations.

The volume of the eyes was calculated from the weights on the assumption that the specific gravity of the eye was 1.

Results.—The concentrations of ascorbic acid in the aqueous humor and the blood of the rabbits are plotted in figure 1 as a function of the animal's age. It is apparent from the graph that the concentration of ascorbic acid increases abruptly, starting about the ninth day after birth. The concentration in the blood during the same interval remained the same, varying between zero and 2 mg. per hundred cubic centimeters.

Because of low concentration of ascorbic acid and the small quantity of aqueous humor which could be obtained from eyes of rabbits less than 1 week old, it was not possible to determine accurately the concentration of the vitamin in

From the Pathology Laboratories of the Massachusetts Eye and Ear Infirmary, and the Harvard Medical School.

1. Kinsey, V. E., and Grant, W. M.: The Mechanism of Aqueous Humor Formation Inferred from Chemical Studies on Blood–Aqueous Humor Dynamics, J. Gen. Physiol. **26**:131, 1942.

2. Terry, T. L.: Ocular Maldevelopment in Extremely Premature Infants, J. A. M. A. **128**:582 (June 23) 1945.

3. Friedenwald, J. S.; Buschke, W., and Michel, H. O.: Role of Ascorbic Acid (Vitamin C) in Secretion of Intraocular Fluid, Arch. Ophth. **29**:535 (April) 1943.

4. Mindlin, R. L., and Butler, A. M.: The Determination of Ascorbic Acid in Plasma: A Macromethod and Micromethod, J. Biol. Chem. **122**:673, 1938.

the aqueous humor of these eyes. However, it is possible to say that in none of the 3 instances in which attempts were made to analyze these samples did the ascorbic acid concentration exceed 3 mg. per hundred cubic centimeters. Accordingly, it appears certain that the ascorbic acid concentration in the aqueous humor of the eyes of rabbits younger than 8 days of age does not differ significantly from the concentration in the blood.

It is noteworthy that the time at which the ascorbic acid concentration begins to increase coincides with the average age at which the adhesions between the eyelids break down and the eyes open.

In order to determine whether there is a postnatal rise of ascorbic acid in the aqueous humor of other species, a few preliminary determinations were made on the aqueous humor–blood ratio for ascorbic acid in the guinea pig. The concentration of the ascorbic acid was determined in the aqueous humor of 3 guinea pigs on the day of their birth and in the aqueous humor of 2 guinea pigs 8 days old. These concentrations were compared with those found in

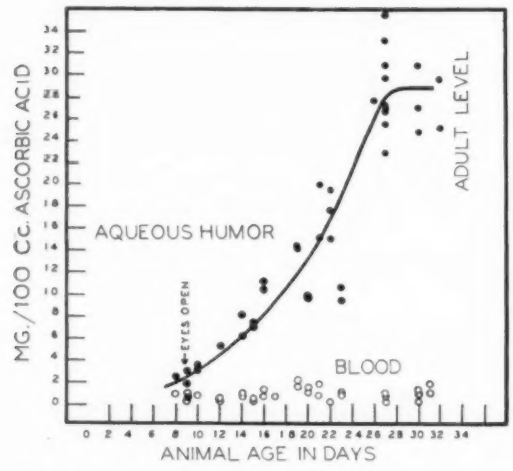

Fig. 1.—Chart showing the concentration of ascorbic acid in the aqueous humor and in the blood of rabbits of various ages.

the mother animals. No consistent differences in the young and in the adult animals were encountered.

With the object of determining the rate at which other constituents of the aqueous humor increase after birth, the total volume of aqueous humor was determined in the eyes of rabbits of various ages. Since the eyes are growing during this period, the volumes of the eyes were also measured, so that the relative increase in the volume of aqueous humor over that expected from the growth of the eye could be indicated by plotting the ratio of the volume of the aqueous humor to the total volume of the eye.

The volume of aqueous humor, the volume of the eye and the ratio of the former to the latter are all plotted in figure 2. It will be seen that both volumes increase linearly with the age of the animal from the first day of life; however, the volume of the aqueous humor is essentially zero at birth, whereas the volume of the eye is approximately 250 cu. mm. Thus, unlike the ascorbic acid content of aqueous humor, the ratio

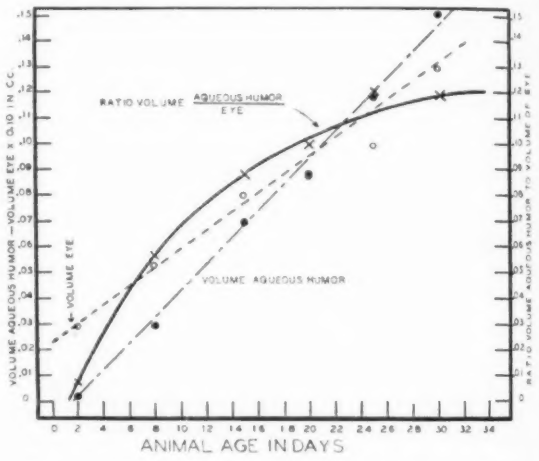

Fig. 2.—Chart showing the volume of aqueous humor, the volume of the eye and the ratio of these volumes in rabbits of various ages.

of the volumes increases fairly rapidly during the first few days but, like the former, approaches a constant value when the animals are about 30 days old.[5]

COMMENT

The significance of the results here described may be interpreted on the basis of the studies of Friedenwald and co-workers.[6] They showed that the energy for secretion could be derived from differences in the oxidation-reduction potential between the epithelium and the stroma of the ciliary body. This difference in potential is due to different enzyme systems operating in the epithelium and in the stroma; the cytochrome oxidase system predominates in the epithelium

5. The ratio of the volume of aqueous humor to the volume of the eye in adult rabbits of another series was found to be 0.105: (Kinsey, V. E.; Grant, M., and Cogan, D. G.: Water Movement and Eye, Arch. Ophth. **27**:242 [Feb.] 1942).

6. Friedenwald, J. S., and Stiehler, R. D.: Circulation of the Aqueous: VII. A Mechanism of Secretion of the Intraocular Fluid, Arch. Ophth. **20**:761 (Nov.) 1938. Friedenwald and associates.[3]

and the dehydrogenase system in the stroma. Among the identified mediators of this system is vitamin C, which, according to Friedenwald, in addition to its function as a carrier of electrons to the epithelium, appears to be secreted into the anterior chamber.[3]

From the analyses reported on in the present paper it would appear therefore that secretion of ascorbic acid into the aqueous humor of the rabbit does not begin until approximately nine days after birth.

While the ratio of the concentration of ascorbic acid in the aqueous humor to that in the blood in guinea pigs is much lower than in rabbits, Friedenwald's experiments indicate that in general the function of ascorbic acid is the same in the two species. The few analyses which we performed on the eyes of guinea pigs suggest that secretion of this substance begins before birth. This is consistent with the well known general precociousness of this animal at birth.

Several interpretations are possible to account for the observation that the ratio of the volume of aqueous humor to the volume of the eye increases immediately after birth whereas the ascorbic acid concentration does not begin to increase until nine days later. For example, it may be assumed either that the rapid initial increase in volume is due to fluid entering the anterior chamber as a result of an ultrafiltration process or that secretion of other constituents precedes that of ascorbic acid. Until further studies have been made, no means of distinguishing between the two explanations is apparent.

A discussion of the significance of the observations reported here with respect to early intraocular nutrition will be deferred until histologic sections are available showing the state of morphologic differentiation and maturation of the eye as a whole.

SUMMARY

The ascorbic acid contents of the aqueous humor and blood were measured in a series of rabbits from the time of birth until the age of 32 days. The concentration of ascorbic acid in the aqueous humor was found to increase rapidly after the ninth day, reaching the level found in adult animals approximately eighteen days later, whereas the concentration in the blood remained essentially constant. The concentration of ascorbic acid in guinea pigs at birth did not differ significantly from that in the adult animals.

Both the volume of the aqueous humor and the volume of the eye increased linearly with age from birth. The ratio of the volume of aqueous humor to the volume of the whole eye increased rapidly from the first day of life, to reach a constant value at the same time (27 days of age) that the ascorbic acid concentration reached a maximum.

Mrs. Rhoda Burden gave technical assistance in carrying out these investigations.

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RELATION OF VISUAL ACUITY TO MYOPIA

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Although data demonstrating the relationship of visual acuity to myopia do exist, no systematic observations over a wide range of degrees of myopia have been made. For example, Laurance and Wood [1] give the following tabulation for degrees of myopia ranging from 0.25 to 2.50 D.:

Myopia, D....	2.50	2.00	1.50	1.00	0.75	0.50	0.25
Visual acuity..	6/60	6/36	6/24	6/18	6/12	6/9	6/6

In a study of 1,860 white school children in Washington, D. C., Kempf and associates [2] found 123 with myopia ranging in degree from 0.25 to 11.00 D. The relationship of myopia to visual acuity is presented graphically (fig. 13 of that paper) by a curve which becomes asymptotic to the myopia axis at a visual acuity of 20/200. These highly improbable results are due in part to the fact that only 2 of the 123 subjects had myopia of greater degree than 4.50 D. and the entire asymptotic tail of the curve, extending from myopia of 3.50 D. to myopia of 11.00 D., was determined by these two points. Also of some significance in explaining these results is the fact that the largest letter on the standard Snellen chart is the 20/200 letter.

In the present study the degree of myopia and the visual acuity were determined for 64 eyes with degrees of myopia ranging from 0.50 to 13.50 D. The mean measure of myopia was 2.37 D., and the degree for 50 per cent of the subjects lay between 1.25 and 5.00 D.

SUBJECTS AND METHODS

The subjects were students at the University of California who had consulted the optometry clinic concerning their myopia. Because the armed services reject candidates having poor visual acuity, and also because claims have been made that myopia is curable, the subjects were eager to participate in any work which might throw light on their condition. All the observers who had myopia of greater degree than 2.00 D. were wearing correcting lenses.

The measure of the subject's myopia was taken as the weakest concave lens which gave a maximum visual acuity. This was checked by the use of a retinoscope. If a relatively small amount of astigmatism was present (less than 0.50 D. cyl. in the lower corrections, less than 1.00 D. cyl. in the medium corrections and less than 2.00 D. cyl. in the high corrections), the subject was accepted, and the "equivalent sphere" was used as the measure of his myopia. The "equivalent sphere" is the average of the spherical lenses required in the two major meridians. For example, if the correction was -2.00 D. sph. $\bigcirc$ -0.50 D. cyl., axis 90, then the power in the 90 degree meridian would be -2.00 D., while that in the 180 degree meridian would be -2.50 D. The "equivalent sphere" would be the average of these two, or -2.25 D.

Visual acuities from 20/10 to 20/800 were measured by the Clason visual acuity meter. This instrument, which projects letters on a screen 20 feet (6 meters) from the observer, is so constructed that the letters remain in focus while their size is varied by moving the lens system forward or backward along a track. The subject was instructed to hold a card over one eye and to watch the screen. The size of the letters at the beginning of the test was below the observer's threshold. The size of the letters presented was now increased until the subject thought he could read them. At this point he asked the operator to stop and reported what he saw on the screen. If he could read more than half the letters of a given line, this line was accepted as the measure of his visual acuity. If he could not read the letters at this point, the size of the letters presented was increased slightly, and he was allowed to try again. This procedure was repeated until the subject could read more than half the letters of any given line. The subject was instructed to try to see the letters but not to squint his eyelids, rather to keep his eyes wide open.

Visual acuities lower than 20/800 were measured by allowing the subject to approach a regular Snellen chart. Thus, if a subject walked to within 4 feet (120 cm.) of the chart in order to read the 20/200 letter, his visual acuity was recorded as 4/200, or 20/1,000. This reading was then checked by allowing him to continue to approach the chart until he could read the next lower line. Thus, if he now approached to within 2 feet (60 cm.) to read the 20/100 letters, his vision was recorded as 2/100, or 20/1,000. If the two readings were not the same, a third reading was taken and the three averaged. In this method of testing the subject was again instructed to keep his eyes wide open and to approach the chart until he thought he could read a given line, at which time he was to stop walking and

From the Vision Laboratory, Department of Physiology, Stanford University, Calif., and the School of Optometry, University of California, Berkeley, Calif.

Help in the statistical analysis of the data was given by Prof. Frank W. Weymouth, of the Department of Physiology, Stanford University.

1. Laurance, L., and Wood, H. O.: Visual Optics and Sight Testing, ed. 4, Chicago, Chicago Medical Book Company, 1936.

2. Kempf, G. A.; Collins, S. D., and Jarman, B. L.: Refractive Errors in Eyes of Children as Determined by Retinoscopic Examination with a Cycloplegic, Public Health Bulletin 182, Washington, D. C., Government Printing Office, 1928.

try to read the chart. He was cautioned against overstepping and was advised to attempt to read the letters at the farthest point possible.

RESULTS

The visual acuity and the degree of myopia were determined in the manner described for 64 eyes. Figure 1 is a scatter diagram on a

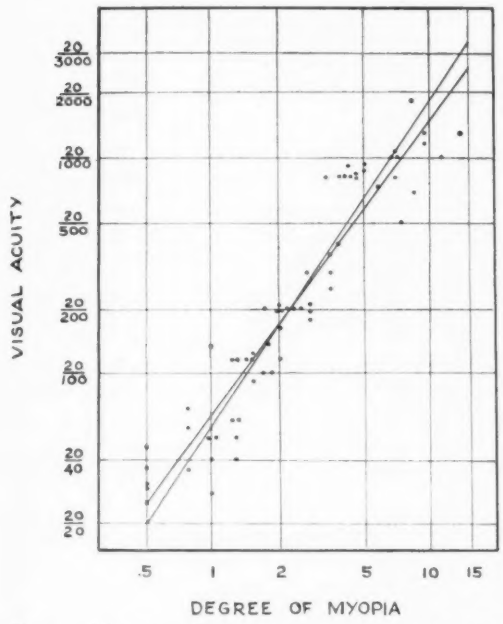

Fig. 1.—Visual acuity and degree of myopia plotted on a double logarithmic scale.

double logarithmic scale, showing the relation between degree of myopia and visual acuity. The correlation coefficient for the relationship of the logarithm of degree of myopia to the logarithm of visual acuity is + 0.95. Figure 1 also shows the regression line of the logarithm of degree of myopia on the logarithm of visual acuity and the regression line of the logarithm of visual acuity on the logarithm of degree of myopia. Each regression line has been plotted separately (figs. 2 and 3) together with confidence limits of 50 and 95 per cent. If the regression lines are used for prediction, the 50 per cent confidence limits indicate the range within which one may predict and be correct 50 per cent of the time. Similarly, if one predicts that a given point will fall within the range of the 95 per cent confidence limits, the prediction will be correct 95 per cent of the time. The 50 per cent confidence limits lie at $\pm$ 0.6745 times, and the 95 per cent confidence limits at $\pm$ 1.96 times, the standard error of estimate.

The formulas for the two regression lines are:

$$\log D = 1.360 \log M + 1.817 \quad (1)$$

$$\log M = 0.663 \log D - 1.169 \quad (2)$$

M in formulas 1 and 2 represents the degree of myopia. D is the denominator of the Snellen fraction, and visual acuity equals 20/D. Thus, a value of 80 for D represents a visual acuity of 20/80, while a value of 400 for D represents a visual acuity of 20/400.

The formulas may also be expressed as follows:

$$D = 65.62\, M^{1.360} \quad (1a)$$

$$M = \frac{D^{0.663}}{14.76} \quad (2a)$$

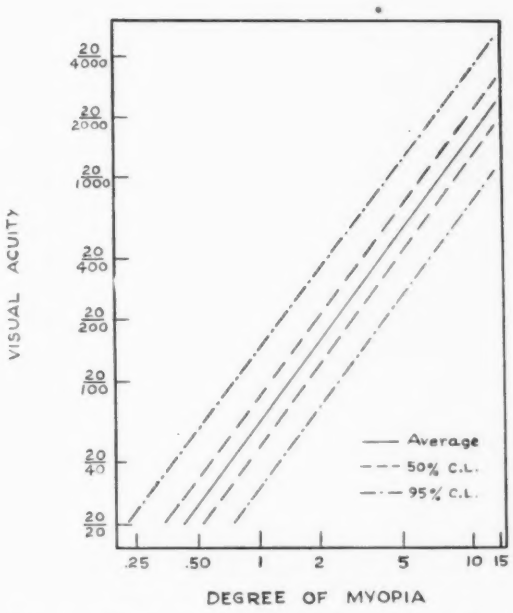

Fig. 2.—Visual acuities plotted for various degrees of myopia.

TABLE 1.—*Approximate Visual Acuities for Various Degrees of Myopia*

Myopia, D.	Visual Acuity				
		50% Confidence Limits		95% Confidence Limits	
	Average	Maximum	Minimum	Maximum	Minimum
0.50	20/25	20/20	20/35	20/13	20/60
1.00	20/65	20/50	20/85	20/30	20/150
1.50	20/110	20/85	20/150	20/50	20/250
2.00	20/165	20/130	20/220	20/75	20/380
2.50	20/215	20/175	20/300	20/100	20/500
3.00	20/285	20/225	20/380	20/130	20/650
3.50	20/350	20/280	20/475	20/165	20/800
4.00	20/420	20/340	20/575	20/200	20/950
4.50	20/500	20/390	20/675	20/230	20/1100
5.00	20/565	20/450	20/780	20/265	20/1300
6.00	20/725	20/575	20/1000	20/340	20/1650
7.00	20/900	20/700	20/1225	20/420	20/2025
8.00	20/1100	20/850	20/1475	20/500	20/2425
9.00	20/1300	20/1000	20/1750	20/600	20/2875
10.00	20/1500	20/1150	20/2000	20/700	20/3325
12.00	20/1900	20/1500	20/2600	20/940	20/4200
14.00	20/2400	20/1850	20/3000	20/1230	20/5150

Table 1 contains the data presented graphically in figure 2. This table gives the expected visual acuity for a given degree of myopia and was calculated from equation 1. Table 2, containing the data graphically presented in figure 3, was calculated from equation 2 and shows the expected degree of myopia for a given visual acuity.

TABLE 2.—*Approximate Degrees of Myopia for Various Visual Acuities*

Visual Acuity	Myopia (Diopters)				
		50% Confidence Limits		95% Confidence Limits	
	Average	Maximum	Minimum	Maximum	Minimum
20/30	0.62	0.75	0.50	1.12	0.37
20/40	0.75	1.00	0.62	1.37	0.50
20/50	0.87	1.12	0.75	1.50	0.50
20/60	1.00	1.25	0.87	1.75	0.62
20/80	1.25	1.50	1.00	2.00	0.75
20/100	1.50	1.75	1.25	2.37	0.87
20/150	1.87	2.25	1.62	3.00	1.12
20/200	2.25	2.75	2.00	3.75	1.37
20/300	3.00	3.50	2.50	5.00	1.75
20/400	3.50	4.25	3.00	6.00	2.00
20/500	4.00	5.00	3.50	7.00	2.37
20/600	4.50	5.62	4.00	7.75	2.75
20/700	5.00	6.25	4.50	8.50	3.00
20/800	5.50	6.87	4.87	9.37	3.25
20/900	6.00	7.50	5.25	10.00	3.50
20/1000	6.50	8.00	5.50	10.62	3.75
20/1500	8.50	10.50	7.00	13.75	5.00
20/2000	10.25	12.50	8.25	16.50	6.00

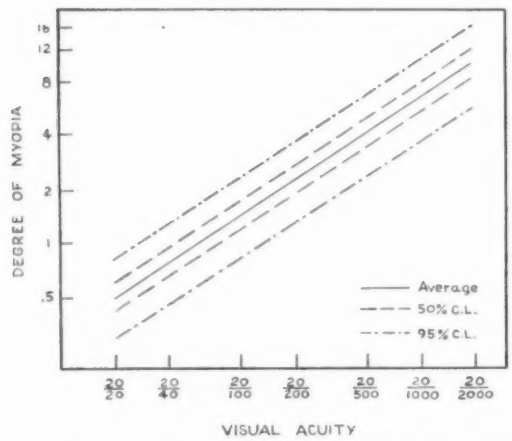

Fig. 3.—Degrees of myopia plotted for various visual acuities.

These equations and tables are valid only for subjects similar in age to those included in this study, for similar methods of determining visual acuity and for degrees of myopia ranging from 0.50 to 15.00 D. That the equations will not hold in cases of emmetropia (with degrees of myopia below the range just mentioned) is demonstrated by substituting 0.00 for M in equation 1a. This would give a value of 0 for D, or a visual acuity of 20/0, which is of course impossible.

CONCLUSIONS

For the homogeneous group described, visual acuity decreases in an orderly fashion as the degree of myopia increases. The relation is not linear but, rather, one fitting an exponential equation. From the tables, equations or graphs presented, it is possible to predict within certain limits either the visual acuity or the degree of myopia which may be expected if the other value is known. The following practical applications may be made of the data:

1. In clinical refraction, visual acuity is usually obtained early in the examination. Reference to table 2 will yield the expected degree of myopia and the confidence limits. If further testing (skiametric and subjective methods) shows a degree of myopia in excess of the maximum for 95 per cent confidence limits, one should suspect that the correction being determined is in fact an overcorrection of the myopia.

This is not a new idea. The old time spectacle peddler and early refractionist, who depended solely on subjective methods, used visual acuity as a determinant of the starting point of his trial and error procedure. Also, the well known rule of thumb that "minus lens may be safely added to the correction so long as each 0.25 D of concave sphere increases acuity by one line of Snellen type," is based on this same idea.

2. The use of the visual acuity fraction as a determinant of fitness for military service by the United States Army has led to many strange situations. Although many persons with 3.00 or 4.00 D. of myopia have received selective service classifications exempting them from service on the basis of their myopia, others with as much as 12.00 D. of myopia have been accepted by the Army. The use of the data contained in this paper will not rectify this situation as completely as would the adoption of the refractive state as a standard for selection, but it would tend to reduce the range of myopia found in acceptable candidates.

Since the minimum visual acuity for the Army is 20/400, reference to table 2 gives minimum and maximum degrees of myopia for 95 per cent confidence limits at 2.00 and 6.00 D. Thus, one may say that any person with myopia of 6.00 D. or more who asserts that he can read the 20/400 letters is probably squinting or memorizing the chart. Conversely, any applicant who manifests less than 2.00 D of myopia and claims inability to read the chart solely on the basis of his myopia is probably malingering. Table 1, in which may be found the expected performance for persons with a given degree of myopia, would be a fur-

ther aid in detecting malingering, squinting the eyelids or memorization of the charts.

Visual acuity is also utilized in determining employability in certain industries, and the industral problems lend themselves to a similar analysis and solution. If a certain task requires 20/40 vision, reference to table 2 shows the minimum degree of myopia to be 0.50 D. Thus, all employees on this job who manifest more than 0.50 D. of myopia should wear correcting lenses despite the fact that by memorization or squinting they are able to read the required line of the chart.

SUMMARY

Visual acuity and the degree of myopia were determined for 64 eyes. The subjects were all college students whose myopia ranged from 0.50 to 13.50 D.

Plotting the logarithm of visual acuity on the logarithm of the degree of myopia gives a coefficient of correlation of $+0.95$.

Confidence limits were determined for the regression lines and tables set up for predicting either the degree of myopia or the visual acuity if the other is known.

These tables have value in refraction in guarding against an overcorrection of the myopia.

The data presented would also be of value in detecting malingering, squinting of the eyelids or memorization of the charts in testing personnel for military service or for employment in industry.

Department of Physiology, Stanford University.

MELANOMA OF THE SKIN WITH INTRAOCULAR AND ORBITAL METASTASES

REPORT OF A CASE

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NEW YORK

It has been stated by Virchow that organs which are a frequent site of origin of primary cancer are prone not to have metastatic growths. Although it is now recognized that there are many exceptions to this statement, the rule seems to apply with peculiar force to the melanotic malignant growths of the eye. Most of the melanomas of this organ are known to be primary in the choroid. Only a few have been reported as metastasizing to the eye from other sites of origin. Recently there came under observation at City Hospital a patient with the unusual occurrence of a melanotic tumor arising in the skin and producing intraocular metastases. In addition, his case presented the unique feature of metastasis to the orbital tissue. This case forms the basis of the present report.

REPORT OF CASE

History.—A white man aged 68 was admitted to the first medical division, service of Dr. Henry Marks, City Hospital, Welfare Island, on July 27, 1944, complaining of axillary pain, of five months' duration. Two years previously, a painful "strawberry" mass appeared in the skin of the right posterior wall of the chest. The growth was excised surgically, an area about 2 cm. in diameter being removed. The histologic diagnosis is not available. The patient remained in good health for eighteen months. Then, at the site of the previous operation, another mass appeared, which was very painful; its appearance was soon followed by severe pain and swelling in the axilla. The axillary mass was removed, mainly for relief of pain. Histologic examination showed metastatic malignant melanoma of the lymphatic nodes. The patient also had pain in the tibia, both at rest and while walking. Shortly after the second operation, widely scattered subcutaneous masses developed over the entire body. Some nodules ulcerated through the skin and were very painful. More recently, there had been increasing weakness, considerable loss of weight and progressive loss of vision. There were no other data pertinent to his condition.

Physical Examination.—The patient appeared extremely anemic and chronically ill. He complained chiefly of persistent pain in the right axilla. Scattered over the entire body were innumerable masses, ranging in size from 1 mm. to 1 cm. Some were subcutaneous and freely movable, and others were fixed to the skin; the largest tended to ulcerate and were reddish violaceous and painful. The scar of the first operation was bright violaceous and was surrounded by many small masses. In the axillary scar were many blue-black nodules. The left axillary lymph nodes were enlarged and painful. The chest was diffusely painful to percussion. The temperature was normal, 98.6 F.; the pulse was somewhat rapid, 90 per minute, and the respiratory rate was 20 per minute. The urine contained melanin. There were no other observations of note in the general examination.

Fig. 1.—*A*, metastatic mass in the orbit attached to the normal optic nerve of the left eye; *B*, choroidal lesions beneath the pigment epithelium, and *C*, second choroidal focus with melanotic cells.

Ophthalmologic examination, made two days before the patient's death by Dr. Alfred Kestenbaum, showed the following condition: The pupils reacted normally

Read before the New York Society for Clinical Ophthalmology, Feb. 5, 1945.

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to light and in accommodation. The left pupil was irregular in shape, owing to a raised yellow nodule on the nasal side of the iris.

Right Eye: The retina was detached on the nasal side to an elevation of 10 D. and was gray-green. A corresponding loss of the temporal field was demonstrated by the confrontation test.

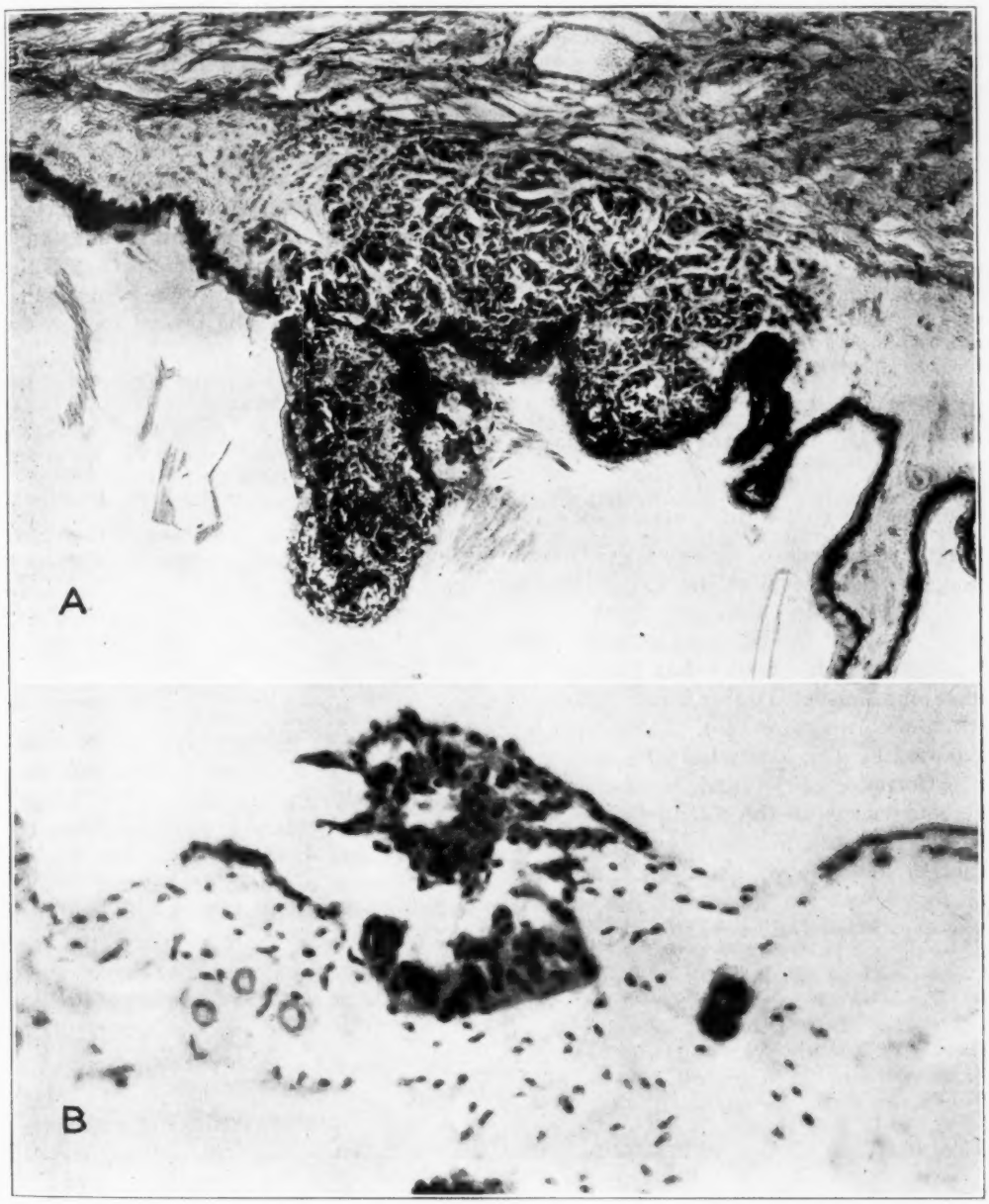

Fig. 2.—*A*, metastatic lesions in the ciliary body, and *B*, metastatic lesion in the iris.

Left Eye: A detachment similar to that observed in the right eye was seen on the temporal side.

Course.—The patient failed rapidly and died on the sixth day in the hospital, of cardiac failure and terminal pneumonia.

Autopsy (performed two and a half hours after death).—There were widespread metastases in the organs, only the testes and the left epididymis being uninvolved. The ribs, vertebrae and sternum had a diffuse jet black color. The base of the skull was similarly discolored. The vault had scattered areas of pigmentation. The other bones were not examined. The cerebrum and cerebellum, dura and leptomeninges had innumerable tumors. One pigmented mass was present in the pituitary. A smear of the heart blood showed melanoma cells.

Only the left eye was removed. In the orbital fat on the nasal side was a roughly globular white mass, lightly attached to the optic nerve by fine fibrous adhesions (fig. 1*A*). Externally the eye was normal. On section, a gray-black mass was seen to occupy the posterior part of the globe about the nerve head, reach-

ing the equator of the bulb on the midtemporal and inferior sides. The tumor was situated in and completely replaced the choroid. It consisted of groups of polyhydral cells in a well vascularized connective tissue. The nuclei were hyperchromatic and varied greatly in size, shape and staining reaction. Cytoplasmic melanin was usually abundant; a few cells appeared devoid of it. Over the summit of the mass, the pigmentary layer of the retina was closely apposed (fig. 1*B*), the nucleated layers being detached by a slight amount of subretinal fluid. At the reflected edges of the tumor the retina was not detached. Extending anteriorly within the choroid, as far as the ora serrata, small masses of melanotic cells were present (fig. 1*C*). The ciliary body had areas of diffuse involvement by similar pigmented cells (fig. 2*A*). In the iris, several similar small masses were situated beneath the anterior limiting membrane (fig. 2*B*).

The orbital mass presented a slightly different appearance. The malignant cells tended to be larger, more separated and more anaplastic and had much less pigment than those in the bulb. The individual nests were surrounded by a fine membrane, similar to the basement membrane of a gland and producing a close resemblance to gland formation. The optic nerve was not invaded.

Diagnosis.—The diagnosis was primary malignant melanoma of the skin with extensive metastases, including intraocular and orbital foci.

COMMENT

Metastatic melanomas of the eye are extremely uncommon. In a review of the medical literature only 7 acceptable cases were found. The first was reported by Schiess-Gefnuseus and Roth,[1] in 1879. This report was followed by the communications of Lauber[2] and Adamük,[3] which dealt with the same case. Later were the cases reported by Ten Doesschaate,[4] Cordes and Horner,[5] Corrado,[6] Fry[7] and Kreibig.[8] The data are summarized in the accompanying table.

In Brömser's[9] case histologic examination of the tumor of the skin, which he considered the primary focus, was not made, and his conclusion was questioned by Fuchs.[10] The case of Pflüger[11] may be authentic, but complete confirmation is lacking.

According to de Schweinitz,[12] tumors of the eye of metastatic nature are characterized by certain features. They tend to be multiple, flat and bilateral. Certain areas of the eye are usually affected. The tissues surrounding the foci are comparatively healthy. The neighboring blood vessels frequently have malignant cells in the lumen. The last criterion must be accepted with some reservation, since this is a feature even of primary malignant melanoma and is also displayed by some carcinomas.

The present case presents many features displayed by the cases previously reported. The site of origin was the skin, similar to that in 6 of the 7 cases in the literature. All the characteristics mentioned by de Schweinitz were likewise present. The orbital metastasis found in our case is a unique feature, not previously reported with melanoma. Willis[13] cited 3 instances of secondary growth involving the orbit and reported 1 of his own, but none of the tumors were of melanotic nature. The cases reviewed suggest that, although most melanotic tumors of the eye are primary, the possibility should be borne in mind that they are secondary and careful search should be conducted for other primary sites and for evidence of multiple metastases.

Data on Cases of Metastatic Melanoma of the Eye

Author	Site of Origin	Sites of Metastasis
Schless-Gemuseus and Roth,[1] 1879	Skin	Papilla and optic nerve
Lauber,[2] 1907 Adamük,[3] 1909	Lids and conjunctiva	Uveal tract
Ten Doesschaate,[4] 1921	Opposite eye	Uveal tract
Cordes and Horner,[5] 1930	Skin	Right and left iris; both ciliary bodies; optic nerve
Corrado,[6] 1931	Skin	Ciliary body
Fry,[7] 1933	Skin	Choroid
Kreibig,[8] 1935	Skin	Choroid
Lisa and Givner, 1945	Skin	Choroid; ciliary body; iris; orbit

SUMMARY

A case of primary malignant melanoma of the skin with intraocular and orbital metastases is reported. The literature is reviewed.

108 East Sixty-Sixth Street.

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CONGENITAL BILATERAL SUBLUXATION OF THE LENS

REPORT OF A FAMILY

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Partial dislocation of the lens presents a definite problem both for the patient and for the surgeon. Recently, Clapp [1] reported on a family with ectopic lenses in which 8 out of 11 children showed this defect. Burch [2] gave a rather complete report of cases of ectopia of the lens, in some of which the additional feature of arachnodactyly was presented. Attempts to improve vision in cases of partial dislocation of the lens frequently have met with failure. Clarke [3] expressed the opinion that the best treatment was removal of the lens. This usually involves the use of a loop, with more or less loss of vitreous, as pointed out by Duke-Elder.[4] Knapp [5] stated the belief that the most satisfactory way to remove the lens is by needling—two needles being used, one to impale the lens and the other to produce a rent in its capsule. He stressed the fact that the lens capsule is singularly difficult to puncture, being easily stretched, and that the lenticular material is only slowly absorbed.

The following case of bilateral subluxation of the lens is of interest because of the family history.

D. G., a 6 year old girl, was first seen on June 27, 1944. The patient was in a sight-saving class at that time and was brought for examination in an effort to learn whether vision might be improved.

The child was rather thin but showed no evidence of arachnodactyly. Vision was 10/200 in the right eye and 3/200 in the left eye. External examination showed that the eyes were normal except for exophoria of 14 D. and a near point of convergence of 120 mm.

The pupillary reaction was normal, but iridodialysis was present in both eyes. The intraocular tension appeared normal on palpation. Both lenses showed subluxation, with displacement upward and inward. With the pupils dilated the zonular fibers were seen to be in place below. The lenses were transparent and showed a few fine pigment granules on their anterior surfaces. Examination of the fundus was unsatisfactory even when the pupils were dilated, owing to the position of the lenses. Retinoscopic examination was impossible, and manifest refraction did not result in improvement of vision.

The past history was without significance. Birth had been at full term, and there was no evidence of developmental anomalies other than those found in the eyes.

Operation was performed on July 10, 1944, needling being done on the right eye, with the two needle technic. As little apparent change was produced, the procedure was repeated on Oct. 16, 1944. Since, the lens has become progressively more opaque and is beginning to shrink.

Family History.—The family history is of extreme interest.

Maternal Grandfather: M. M. was seen in the Vanderbilt Clinic on April 17, 1935, at the age of 56. He gave a history of having been struck in the right eye twenty days before, with resulting loss of vision. Vision had always been poor in both eyes. On examination, vision was 20/200 in the right eye and was limited to counting fingers at 3 inches (7.5 cm.) in the left eye. The right eye was normal externally, and tension was 25 mm. Schiøtz. The lens was partially dislocated downward, and the zonular fibers were visible with the corneal microscope. The left eye was congested, with a tension of 38 mm. Schiøtz. The lens was dislocated and lay in the anterior chamber, and, with the patient lying on his back, it would recede through the dilated pupil. On April 19, 1935 a loop extraction was done without iridectomy. After this, with a correction of + 1.50 D. sph. ⊃ + 5.00 D. cyl., ax 8, vision was 20/50+ in the left eye (an addition of a + 3.00 D. sph. was given for near vision).

On Nov. 26, 1936 a preliminary iridectomy was done on the right eye, with uneventful recovery. Examination on Jan. 13, 1937 showed that the lens had settled back into the vitreous. No operation was attempted. Vision was 20/30—2 in the right eye, with a correction of + 6.25 D. sph. ⊃ + 1.50 D. cyl., ax 180. An addition of a + 3.00 D. sphere gave good near vision.

The patient appeared in the clinic on June 13, 1938, with the complaint of poor vision in the left eye. Vision was 20/40 in the right eye and 20/200 in the left eye. The lower half of the retina of the left eye was found to be detached. On June 20, 1938 a diathermy operation was done on the left eye, with an unsuccessful result. The patient was last seen in March 1939.

Mother: M. G. was seen in the Vanderbilt Clinic on Nov. 13, 1935, at the age of 27. Vision was 20/200 in

From the Institute of Ophthalmology of the Presbyterian Hospital and the Department of Ophthalmology, Columbia University College of Physicians and Surgeons.

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5. Knapp, A.: Operative Treatment of Congenital Subluxation of the Lens, Arch. Ophth. **27**:158-160 (Jan.) 1942.

each eye, and both lenses were partially dislocated upward and nasally. The intraocular tension was normal. On Dec. 2, 1935 a needling was done on the right eye. Recovery was uneventful, but little effect was produced. A through and through needling was done on Feb. 10, 1936. Absorption of the lens progressed slowly. A third needling was done on October 19. After this, the lens gradually disappeared. Vision became 20/50 with a + 10.50 D. sphere.

The patient was last seen on June 24, 1944. Vision in the right eye was then 20/70. There remains some old cortex, but no further operation is desired. Near vision is good with a + 5.00 D. sphere clipped on. The left eye now shows exotropia of 96 D.

Maternal Aunt: A. M. was first seen in the Vanderbilt Clinic on April 17, 1935, at the age of 12. Vision was 20/100 in each eye. Both lenses were partially dislocated upward and nasally. On July 8, 1935 a needling was done on the left eye. Absorption of the capsule was slow. Improvement in vision was obtained with the following correction:

Right eye: + 13.50 D. sph. ⊃ + 1.50 D. cyl., ax 130, vision 20/40.

Left eye: + 14.00 D. sph. vision 20/40.

The patient was last seen on Sept. 16, 1944, when vision was 20/30 in each eye. She wears a + 13.75 D. sphere with each eye. The lens of the right eye is unchanged. The lens of the left eye shows an old membrane and is partly opacified. The intraocular tension is normal. This patient does not like her glasses, nor does she desire further surgical treatment. Manifest refraction revealed no further improvement in vision.

SUMMARY

A case of bilateral subluxation of the lens in a child is presented in which 3 other members of the family had the same disease, three generations being represented. Operation was performed on all these persons. In 1 patient the condition was complicated with glaucoma due to the misplaced lens and by later detachment of the retina in that eye. In all but 1 patient the result of operation was unsatisfactory, and even in this patient complete absorption of the lens has never taken place.

635 West One Hundred and Sixty-Fifth Street (32).

COMPLICATIONS INCIDENT TO SIMPLE INTRACAPSULAR EXTRACTION

SOME PROCEDURES DESIGNED TOWARD PREVENTION

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Intracapsular extraction with preservation of the round pupil is subject to the complications that occur with most cataract operations. Some of these complications are less frequent and less severe; but involvement of the vitreous and iris takes on added significance, and it is the frequency or infrequency of this involvement that determines the success or failure of this procedure.

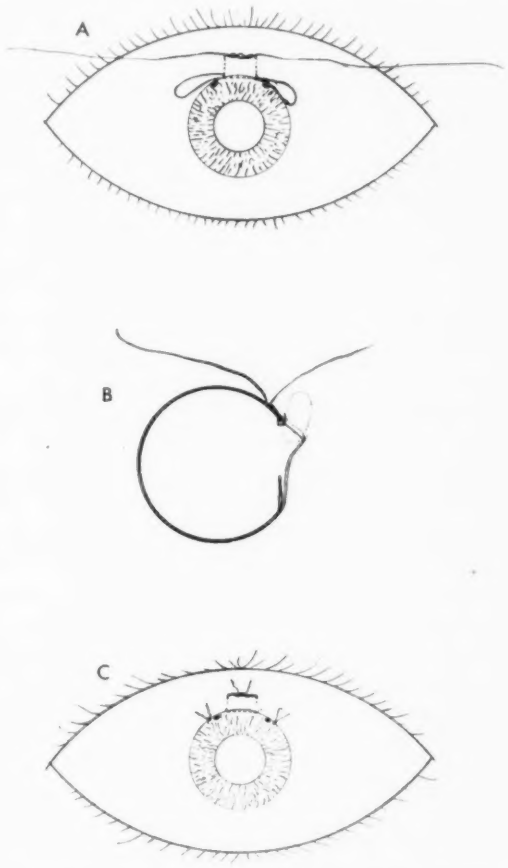
The best defense against the more troublesome complications peculiar to this operation is well placed sutures which insure adequate closure of the incision. In my experience, corneal sutures are more easily placed before opening the globe.

I have used a simple form of mattress, or section halter, suture for the past ten years which I have found very effectual, and I shall describe it briefly.

Prior to section, the globe is fixed with the globe fixation forceps, and a 24 inch (1.56 cm.) silk suture double-armed with Kalt needles is inserted in the superficial stroma of the cornea just below the limbus, above, for a distance of 4 or 5 mm. After the corneal incision, which is made with a narrow conjunctival flap, the suture is completed by grasping the episcleral fibers and the conjunctiva immediately above each end of the corneal suture with the needle point suture forceps, and each needle is passed through deep episcleral tissue and brought out through the conjunctiva 5 or 6 mm. above the incision. A loose horizontal knot is now made. This corneal controlling suture can be tightened, instantly closing the wound at any time, a provision which is invaluable with unruly patients or in the case of sudden prolapse or herniation of the vitreous at or near the end of delivery of the lens. This suture has the value of two sutures in the upper part of the section, but without additional sutures the wound will not immediately become aqueous tight and the chamber will not hold when refilled. I make a practice of using at least two additional corneoscleral or corneoepiscleral sutures at the positions of 10 and 2 o'clock.

RUPTURE OF THE CAPSULE

The delicate procedure of dislocation and removal of the lens in its capsule does not depend on the forceps or the method applied. It depends on delicacy of touch and grasp, which must be acquired by observation and experience before one can grasp the lens and use sufficient traction to rupture the zonule without tearing the capsule.



Sketches illustrating (*A*) conditions just prior to dislocation of the lens, with suture in place and section and iridotomies completed; (*B*) use which may be made of this suture when there is fear of prolapse of the vitreous (the suture is drawn to bring the corneal flap against the emerging lens), and (*C*) completed closure of the wound, with all sutures tied.

Most ruptures of the capsule are caused by pinching the blades too tightly, by grasping an insufficient amount of capsule or by applying too much traction before rupture of the zonule has taken place.

The site for application of the capsule forceps is controversial and must be determined by the individual surgeon. Knapp's [1] well known method of grasping and dislocating the lens has been adopted by many surgeons. Smith [2] stated that to Arnold Knapp belongs the credit of showing the world how to use a capsule forceps properly. Verhoeff,[3] Kirby [4] and others grasp the capsule near the equator, above. Kirby [4] cited several disadvantages of grasping it below.

In my experience, few capsules tear if a wide grasp is made well down behind the iris. This permits traction to be applied to a very limited area. In pressing the Smith hook on the limbus over the point of traction, rupture of the zonule can be started without too much traction or injury to the ciliary body. After rupture of the zonule has begun, the remaining fibers separate easily, but not easily enough to permit pulling the tumbling lens up through the pupil. To avoid tearing the capsule, the major force in the maneuver must be supplied by the hook, with forceps traction as the guiding force. However, when a broken or greatly weakened zonule is indicated by extrusion and reextrusion of the iris immediately after section, it is less hazardous to grasp the lens in its upper third and deliver it head on, sliding the cataract from the eye. If the capsule should rupture before the zonule is broken and a great part of the capsule be removed with the forceps, the nucleus and the lens matter can be expressed and irrigated and removed from the chamber, and the procedure becomes a simple extracapsular extraction. However, after rupture, if the capsular opening is small, with the probability of considerable cortex remaining encased behind the anterior capsule and the contracted pupil, a complete iridectomy should be done, and the lens matter should be removed. Rupture of the capsule after the zonule is broken usually comes toward the end of delivery; and if the hyaloid membrane has not ruptured, the capsule fragments can be carefully removed. If not easily seen, they show up well with gentle irrigation.

1. Knapp, A.: Arch. Ophth. **44**:1, 1915.
2. Smith, H.: Arch. Ophth. **55**:218, 1926.
3. Verhoeff, F. H.: Tr. Am. Ophth. Soc. **25**: 1927.
4. Kirby, D. B.: Am. J. Ophth. **27**:124, 1944.

RUPTURE OF THE HYALOID MEMBRANE AND PROLAPSE OF THE VITREOUS

Rupture of the hyaloid membrane and prolapse of the vitreous call for a complete iridectomy. I am in accord with Kirby [5] and Dunnington [6] in that a complete iridectomy should be done in cases in which loss of vitreous is anticipated. I must admit I have not always followed this rule. In a few cases of fluid vitreous, in which loss of vitreous was small, I endeavored to preserve the round pupil. In most of these cases the results were gratifying. The vitreous receded behind the iris, and the eyes function and appear as well as eyes in which this complication did not occur. In a few other cases the only complication was a decentered pupil, with a thin, transparent, striped band extending through the pupil, over the iris, to the point of incarceration in the section, with practically no interference with vision.

With the round pupil operation, prolapse of the vitreous, no matter how small, is a complication of importance. While a complete iridectomy does not eliminate all the unpleasant sequelae, it greatly reduces the reaction on the part of the iris and the ciliary body.

PROLAPSE OF THE IRIS

A securely closed wound is the greatest defense against prolapse of the iris. I performed this operation for several years without the use of sutures, depending entirely on a large conjunctival flap. I then used conjunctival sutures for a time, but, in my experience, they gave no additional security. Actual prolapse occurred in only 7 per cent of these cases, but in a number of instances there were varying degrees of peripheral incarceration or anterior adhesion of the iris to the wound. This was evident only on close inspection and caused no postoperative interference with healing or function. However, within the last year tension has developed in 2 of these cases, almost ten years after extraction of the lens. Often this adhesion of the iris to the incision is the first step in prolapse and is unavoidable in a number of cases unless the wound is made water tight or aqueous tight and the iris and the cornea are separated by a filled chamber shortly after completion of the operation. After the sutures just described are

5. Kirby, D. B.: Prevention and Handling of Complications Arising During and After Cataract Extraction, Arch. Ophth. **25**:866 (May) 1941.
6. Dunnington, J. H.: New York State J. Med. **44**: 2224, 1944.

tightened and locked and I have made certain that the iris is in position, I fill the chamber with saline solution.

In some cases the patient has had violent nausea or has fallen out of bed without opening the wound; on the other hand, I have found a prolapsed iris when there was no report of disturbance. But if a wound is closed securely enough to hold the chamber, prolapse seldom develops except through direct trauma.

A minor detail, which saves trouble at times, is the position of the iridotomy or the peripheral iridectomy. If the opening is made in the mid-position above, it is often difficult to disengage and unfold the iris from the wound, where it has been folded back during delivery. Placement of the iridotomy at 10 and 2 o'clock, leaving the most dependent part of the iris whole, simplifies the process of stroking the iris down into place and lessens the danger of rupturing the hyaloid membrane.

Another detail which may escape notice is wedging or tucking of a narrow fold of iris into the iris angle during section. This fold is easily freed before dislocation of the lens but is frequently difficult to disengage afterward.

It has been my experience that the earlier the operation for prolapse of the iris is performed, the more easily the tissue is replaced or the more cleanly it is excised, as conditions demand. Secure sutures do not give way, and replacement or excision is not the hazardous procedure of a few years ago.

It is generally agreed that if the prolapsed portion contains vitreous, excision is imperative. If the prolapse is large enough to involve the pupillary margin, a small iridectomy is expedient. However, when the prolapsed portion is small and is easily replaced in the chamber, a small peripheral iridectomy, with an additional suture placed at the site of extrusion, completely restores closure of the wound and the position and action of the pupil.

HEMORRHAGE INTO THE ANTERIOR CHAMBER

Hemorrhage into the anterior chamber is a fairly frequent complication of all cataract operations. In the intracapsular operation, with the iris intact, the possibility of a hemorrhage attaining sufficient size to rupture the wound and then to extrude the iris has to be considered. It is generally agreed that the usual source of the hemorrhage is the episcleral vessels under the conjunctival flap. However, the iris is occasionally the source, particularly in the diabetic patient.

Corneoscleral suturing has materially reduced the frequency and the amount of hemorrhage. McLean,[7] Kirby,[5] Stallard[8] and others have reported fewer hemorrhages with use of the corneoscleral sutures. Suturing has definitely reduced this complication, but I still find hemorrhage in about 10 per cent of cases. However, the majority of such hemorrhages are smaller than formerly. One of the greatest benefits derived from suturing is that even with the large hemorrhages substantial sutures hold fast, the wound does not gape and the blood which escapes from the chamber oozes out slowly so that the iris is practically never carried into the wound. In several cases in which the blood has filled the chamber and elevated the tension, the hemorrhage produced great pain without rupturing the sutures or causing prolapse of the iris.

Removal of sutures is a delicate procedure, and unless it is so regarded hemorrhage and rupture of the wound result. I have found O'Brien's method of lid block most helpful in quieting the extremely nervous patient for removal of sutures. When the lid cannot be closed, there is not the tendency to rotate the eye upward, and the nervousness diminishes greatly.

The treatment of hemorrhages, large and small, is the same and has been well summed up by Dunnington,[6] who stated that the vast majority of patients with this complication need little more than rest, atropine, time and patience.

DELAYED RESTORATION OF THE ANTERIOR CHAMBER

Delayed restoration of the anterior chamber is the result of faulty closure of the wound or inefficient wound toilet.

With the intracapsular operation, the iris must be carefully replaced in the globe before the sutures are tightened. If a capsulotomy has been performed, the lens matter and shreds of capsule must be irrigated and removed from the chamber. If this is carefully done and the sutures are placed into structural tissues, the anterior chamber will be reformed and virtually normal in depth a few hours after operation.

In routine dressing of the wound the morning after operation, I have found extreme shal-

7. McLean, J. M.: New Corneoscleral Suture, Arch. Ophth. **23**:554 (March) 1940.

8. Stallard, H. B.: Brit. J. Ophth. **22**:269, 1938.

lowness or absence of the chamber in less than 0.28 per cent of cases since employing corneoscleral sutures.

In my opinion, the necessity of secure closure of the wound cannot be too strongly stressed. It is one of the most important requirements for success with the round pupil operation. Although this calls for patience and care in the suturing, the results gained are gratifying. Immediate restoration of the chamber is obtained, which virtually eliminates peripheral synechia, prolapse of the iris, adhesions between the hyaloid membrane and the posterior surface of the cornea, epithelization of the anterior chamber and early secondary glaucoma.

55 Park Avenue.

Clinical Notes

INSTRUMENTS FOR USE IN OPERATION FOR CATARACT

An Anterior Chamber Irrigator and Two Forceps Modified for Suturing

FRANKLIN BRACKEN, M.D., NEW YORK

The anterior chamber irrigator has been used by me for ten years. The two forceps are simple modifications of instruments long in use.

ANTERIOR CHAMBER IRRIGATOR

The anterior chamber irrigator is a curved, flattened cannula, slightly thicker than an iris repositor, and is fitted with a rubber bulb. The posterior lip of the cannula is extended slightly to avoid picking up the iris when one is aspirating blood or air from the chamber. Free blood, iris pigment or foreign matter can be irrigated or aspirated from the chamber through a very narrow opening. This instrument may frequently be used as an iris repositor and is indispensable for refilling or inflating the chamber after the cataract incision has been securely closed with sutures.

GLOBE FIXATION FORCEPS

The globe fixation forceps is a fairly heavy instrument. The shafts are widely separated, so that the globe may be grasped in its entirety. Each shaft may be mounted with two teeth, or there may be the usual two and one. These teeth leave the shaft at almost a right angle. They must be very sharp at the point and broaden rapidly, so that they readily penetrate the conjunctiva and engage the sclera without piercing it. Piercing the sclera is most improbable if the needle pressure is always exerted toward a double-toothed shaft.

The chief use of this forceps is in fixing the globe rigidly for the insertion of corneal and scleral sutures prior to section. The globe is grasped with the teeth engaging the sclera temporally and nasally about 6 mm. back of the limbus. By slightly closing the forceps and at the same time pressing backward, the operator can hold the globe very steady. Deep corneal and scleral sutures may be inserted without tearing the conjunctiva, causing conjunctival hemorrhage or in any way marring the field of operation.

CORNEOSCLERAL SUTURE FORCEPS

The corneoscleral suture forceps is not unlike a miniature Elschnig forceps. The round teeth are needle sharp and leave the shaft at an angle of 135 degrees. This forceps enables the operator to grasp the beveled edge of the corneal section and hold it fast without injuring the tissue. The teeth must be needle sharp and set at an angle that enables the operator to pick up the episcleral fibers above the section without pressing the open globe appreciably. The round teeth are an important feature of this instrument, for when teeth are so small they easily cut the tissues if they have sharp edges.

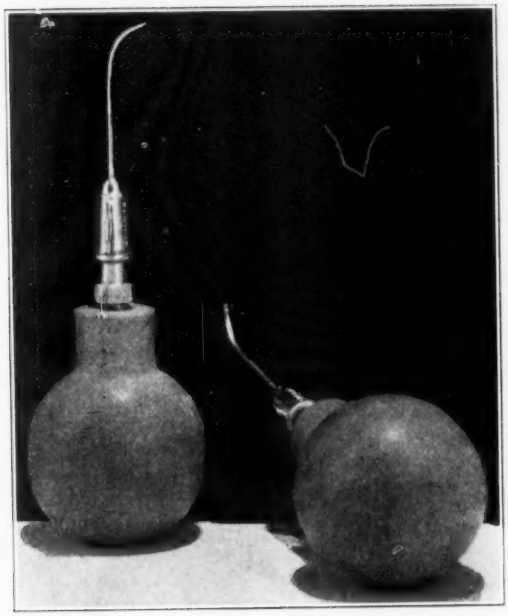


Fig. 1.—Anterior chamber irrigator.

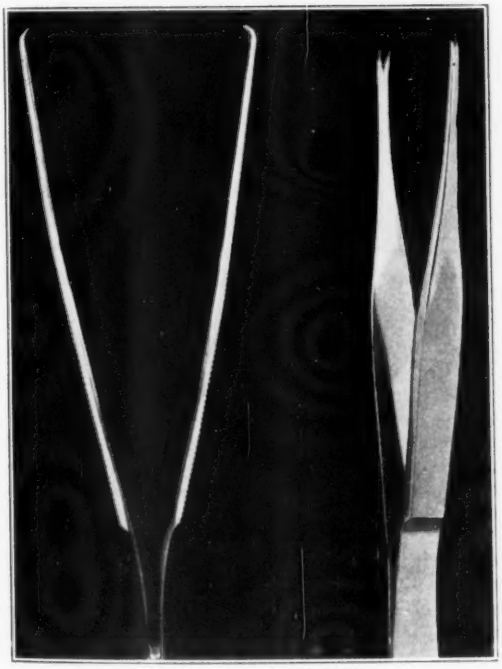


Fig. 2.—Globe fixation forceps.

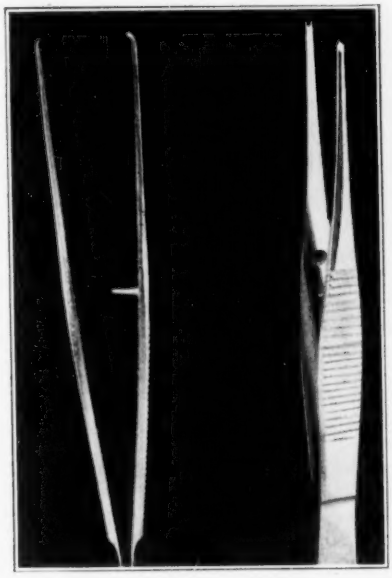


Fig. 3.—Corneoscleral suture forceps.

KERATOCONUS POSTICUS CIRCUMSCRIPTUS

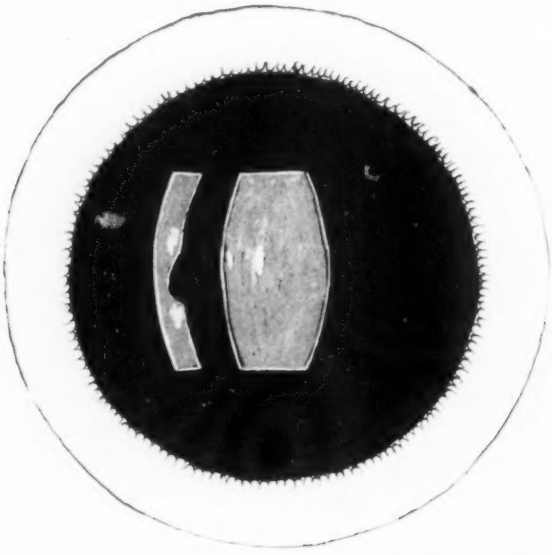
Report of a Case

PHILIP B. GREENE, M.D., SPOKANE, WASH.

There have been 3 cases reported in the English literature [1] and, recently, 2 in the American literature [2] of a condition termed keratoconus posticus circumscriptus. The following case of a patient seen in 1940, which was written up at that time but not published, is of interest in that it may shed some light on the etiology of this minor, and apparently rare, abnormality.

C. E. D., a 52 year old, white business man, consulted me for a refraction on Oct. 5, 1940. His only complaint was difficulty in reading for any length of time. He had worn glasses since childhood, and he volunteered the information that his left eye had never been as good as his right. His health had been excellent for many years. There was no unusual ocular condition in his ancestors. A son, in school, was myopic. No history of trauma was elicited.

Examination disclosed normal conjunctivas and lids. The pupils were round and equal and reacted to light and in accommodation. Focal illumination revealed a small, faint nebula in the left cornea. The right cornea was normal. Vision was 20/200 in each eye without correction. With a —2.50 D. sphere, vision in the right eye was corrected to 20/20. Retinoscopic examination of the left eye showed a refractive error of +0.50 D. sph. ⊂ +0.75 D. cyl., axis 65, and correction gave 20/100 vision.



Optical section through the cornea and lens, showing the arrangement of the opacities in a direct line.

The interesting features of the left eye and the explanation of his poor vision were disclosed by the corneal microscope.

Situated in the middle of the cornea horizontally, but lying somewhat nasally, was the nebula previously mentioned. This was roughly about 3 mm. in diameter and consisted of respersive changes in the substantia propria. Seen in thin optical section and in "block," the cornea was perfectly regular on the anterior surface, but posteriorly it described a sharp anterior curve, with a resultant total thickness of the cornea at this point of about one-third that of the adjacent clear areas. The rest of the cornea was normal. The substantia propria in this area was fairly clear, most of the opacity lying roughly in a ring around it, but the borders of the nebula were not sharp. There was no ectasia of this local thin section.

There was a small anterior subcapsular opacity, about 1 mm. in circumference, slightly nasal to the center of the lens. Under this was a clear area of anterior cortex and adult nucleus. A dense, round, white

1. (*a*) Butler, T. H.: Keratoconus Posticus, Tr. Ophth. Soc. U. Kingdom **50**:551, 1930; (*b*) Two Rare Corneal Conditions, Brit. J. Ophth. **16**:31, 1932; (*c*) Discussion of the Rarer Forms of Keratitis, Tr. Ophth. Soc. U. Kingdom **57**:32, 1937. (*d*) Stallard, in discussion on Butler.[1a] (*e*) Ingram, H. V.: Keratoconus Posticus, Tr. Ophth. Soc. U. Kingdom **56**:263, 1936.

2. (*a*) Leopold, I. H.: Keratoconus Posticus Circumscriptus, Arch. Ophth. **30**:732 (Dec.) 1943. (*b*) Wise, G.: Keratoconus Posticus Circumscriptus, Am. J. Ophth. **27**:1406, 1944.

opacity, somewhat larger, was situated on the face, or in the superficial layers, of the fetal nucleus. The posterior Y suture could be plainly made out, and on the posterior capsule, nasally, could be seen the former site of the hyaloid vessels. Scattered throughout both lenses were fine, punctate, grayish white dots, which were not refractile. The lens of the right eye showed no other opacities except changes in the posterior capsule marking the site of the hyaloid artery. As shown in the diagram, the opacities in the cornea and the lens were in a direct line and gave the impression that such a lesion could have been produced by piercing the cornea and the lens with a needle.

COMMENT

The origin of this undoubtedly congenital condition must remain speculative. The mechanism could be explained by intrauterine inflammation or by an aberration in development.

The time element is fairly definite, as the deepest suture of the lens, the anterior (and the posterior) Y, is formed between the third and the seventh month approximately (Mann). Formation of an adhesion, the result of intrauterine inflammation, between the cornea and the lens at this time, when the anterior chamber is very shallow, might conceivably result in such a condition. This has been said by some authors to occur in cases of anterior lenticonus and of anterior capsular cataract, but this perhaps is not a true explanation, maldevelopment of the anterior zonule explaining the former and weakness in the anterior capsule, possibly a result of the lenticonus, accounting for the latter.

Delayed separation of the lens vesicle from the surface epithelium [3] or, more probably, from the cornea occurring at a later date must be considered. Collins and Parsons [4] described a case of such a nature in a chick, in which the lens vesicle had closed but remained in contact with the cornea. Descemet's membrane had failed to form, and the lens, perfectly developed, remained adherent to the substantia propria. Assuming a similar occurrence in this case, it is conceivable that the adhesion to the cornea ruptured, freeing the lens, but too late to let the normal formation of substantia propria, endothelium and Descemet's membrane from the surrounding mesoblast proceed.

A somewhat similar fetal nuclear opacity, resulting from the adhesion to the lesser circle of the iris, has been described (Conway and Thomson [5]). The adhesion persisted and extended through clear zones of the lens to the iris.

Most congenital opacities of the cornea are ascribed to intrauterine inflammation, and they were termed internal ulcers by von Hippel.[6] Associated inflammation of the anterior uveal tract was described. Absence of Descemet's membrane in the affected area of the cornea is the most characteristic feature. However, Peters [6] suggested as the cause an ectodermal failure or a delay in separation of the lens vesicle, and Collins and Mayou [6] suggested a failure in mesodermal development, resulting in a hiatus in Descemet's membrane. Corneal opacities may be secondary to mechanical rupture of Descemet's membrane in cases of buphthalmos.

As pointed out by Leopold,[2a] Stallard [1d] described the first case of keratoconus posticus circumscriptus, and Butler and Ingram each reported a case, to which have since been added a case by Leopold [2a] and 1 by Wise. This condition is not the same as that which Butler described also under the term keratoconus posticus; the latter is a regular, geometrically precise increase in the curvature of the entire posterior surface of the cornea.

CONCLUSION

A sixth case of keratoconus posticus circumscriptus is described. It is possible that this condition represents a delayed separation of the lens from the cornea and is an aberration in development, although intrauterine inflammation may be considered a possible cause.

517 Old National Bank Building.

3. Hess, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, W. Engelmann, 1911, vol. 11, pt. 9.

4. Collins, E. T., and Parsons, J. H.: Tr. Ophth. Soc. U. Kingdom **23**:244, 1903.

5. Conway, J. A., and Thomson, J. M.: Rare Form of Developmental Cataract, Brit. J. Ophth. **13**:402, 1929.

6. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, p. 1282.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

BLEPHAROPTOSIS

RAYNOLD N. BERKE, M.D.
HACKENSACK, N. J.

I. Definition. Ptosis may be divided into two types:
   1. Pseudoptosis
   2. True ptosis
      A. Acquired
      B. Congenital
      C. Hereditary

II. Etiology
   1. Acquired ptosis
      A. Acute or subacute diseases
      B. Intoxications
      C. Syphilis
      D. Lesions of the brain
      E. Other causes
   2. Congenital ptosis
      A. Abnormal development of the levator muscle
      B. Failure of the nucleus of the third nerve
      C. Evidence to support either theory is to be found in
         a. Postmortem examination
         b. Biopsies
         c. Operative observations
         d. Comparative anatomy
         e. Embryology
   3. Hereditary ptosis

III. Incidence
   1. Of operations for ptosis
   2. Of types of operation

IV. Clinical Anomalies Associated with Ptosis
   1. Abnormal extraocular motility
      A. Weakness of the superior rectus and/or the inferior oblique muscle
      B. Exotropia
      C. Esotropia
      D. External ophthalmoplegia
      E. Duane's retraction syndrome
   2. Abnormal development of the lids
      A. Epicanthus
         a. Superciliary type
         b. Palpebral type
         c. Tarsal type
      B. Blepharophimosis
      C. Abnormal inner canthus
   3. Abnormal innervation of levator, producing jaw-winking (Marcus Gunn phenomenon)
   4. Other abnormal developmental defects

V. Preoperative Study of Ptosis Should Include
   1. Measurement of ptosis
   2. Determination of function of levator
   3. Cocaine test
   4. Extraocular motility
   5. Horizontal length of palpebral fissure
   6. Position of lid fold of each upper lid
   7. Vertical width of upper tarsus
   8. Tests for visual acuity, fusion, diplopia and amblyopia
   9. Sensibility of cornea

VI. Anatomy
   1. Upper lid
   2. Levator muscle
   3. Nerve pathways

VII. Physiology of Upper Lid
   1. Movements of lid with globe
   2. Winking movements
   3. Bell's phenomenon
   4. Blink reflex

VIII. Surgical Treatment of Ptosis
   1. Classification of operations for ptosis
      A. Utilization of frontalis muscle
         a. Surgical variations
            (*a*) Suture material from lid to the frontalis muscle
            (*b*) Skin flaps from lid to frontalis muscle
            (*c*) Strips of orbicularis muscle from lid to frontalis muscle
            (*d*) Levator tendon from lid to frontalis muscle
            (*e*) Strips of fascia lata from lid to frontalis muscle
            (*f*) Flaps of frontalis muscle from brow to upper lid
         b. Comment
            (*a*) Indications and contraindications
            (*b*) Advantages and disadvantages
            (*c*) Results of frontalis type of operation
            (*d*) Reasons for failure
               (*a'*) Stretching of scar bands
               (*b'*) Weakness of frontalis muscle
      B. Utilization of superior rectus muscle
         a. Surgical variations
            (*a*) Strip of superior rectus muscle to tarsus
            (*b*) Tendon of levator to superior rectus muscle
            (*c*) Upper tarsus to superior rectus muscle
            (*d*) Fascia lata from superior rectus muscle to tarsus
            (*e*) Strips of orbicularis from tarsus to superior rectus muscle

b. Comment
(*a*) Indications and contraindications
(*b*) Advantages and disadvantages
(*c*) Results of superior rectus type of operation
(*d*) Reasons for failure
(*a'*) Pulling away of muscle strip
(*b'*) Weakness of superior rectus muscle

C. Utilization of levator palpebrae muscle
a. Surgical variations
(*a*) Shortening of levator muscle through conjunctiva
(*b*) Shortening of levator muscle through skin
(*c*) Excision of part of lid
(*a'*) Resection of tarsus
(*b'*) Resection of orbicularis muscle
(*c'*) Resection of orbicularis muscle and skin
(*d'*) Resection of part of entire thickness of lid
b. Comment
(*a*) Indications and contraindications
(*b*) Advantages and disadvantages
(*c*) Results of shortening levator
(*d*) Reasons for failure
(*a'*) Weak levator muscle
(*b'*) Pulling out of sutures
(*c'*) Resection of Müller's muscle instead of resection of levator muscle or its aponeurosis
(*d'*) Insufficient resection

IX. Criteria for Perfect Operation for Ptosis

X. Summary

XI. Conclusion

DEFINITION

Blepharoptosis is an abnormal drooping of the upper lid and may be divided into pseudoptosis and true ptosis. The former may be caused by an abnormal widening of the palpebral fissure on one side, thus producing an apparent ptosis on the other, or it may be due to disease or increased weight of the lid on the side of the ptosis, such as occurs with edema, elephantiasis, relaxed skin, prolapsed orbital fat, blepharochalasis, trachoma, tumors, endophthalmos, spasm of the orbicularis muscle and neurofibromatosis. True ptosis results from inadequate lifting power of the levator palpebrae muscle and is caused by an abnormality of the levator muscle or of its nerve supply. It may be congenital, acquired or hereditary in origin, unilateral or bilateral, partial or complete.

ETIOLOGY OF PTOSIS

ACQUIRED PTOSIS

Acquired ptosis is usually due to a lesion of the nervous system, to trauma or to disease of the upper lid. According to Wilbrand and Saenger,[1] the chief causes of acquired ptosis are:

1. Acute or subacute diseases, such as diphtheria, influenza, measles, erysipelas, epidemic cerebral meningitis, typhus, pneumonia, acute rheumatism and septicema.

2. Intoxications, such as occur from the coal tar derivatives, lead poisoning and botulism.

3. Syphilis of the upper lid, the orbit, the roots of the third nerve, the nucleus of the third nerve, the supranuclear centers or the cortical centers.

4. Lesions of the brain substance, such as hemorrhage, softening of the brain, abscess formation, meningitis, sinus thrombosis and brain tumor.

5. Other causes, such as myasthenia gravis, trauma, multiple neuritis, Landry's paralysis, polyneuritis, diseases of the orbit or the nasal sinuses, Horner's syndrome, late acquired hereditary ptosis and hysteria.

Acquired ptosis does not become a surgical problem until one is certain that other means of treatment will not effect a cure, and then only if curing the ptosis does not result in diplopia, corneal exposure or ulceration.

CONGENITAL PTOSIS

The cause of congenital ptosis is either a developmental defect of the levator muscle or a lesion of the central nervous system. Because facts to prove either theory are few and inconclusive, there is no unanimity of opinion on this subject, some authors contending that the cause is primarily nervous and others that it is primarily muscular. Evidence to support either theory comes from biopsies, operative observations and postmortem examinations of patients with congenital ptosis. Embryology and comparative anatomy also help to explain the origin of congenital ptosis.

Anatomic and Histologic Studies on Congenital Ptosis.—In 1900 Wilbrand and Saenger,[1] in their monumental work, reviewed the literature on congenital ptosis up to that time. They were able to find the report of only 1 case in which the entire orbital contents had been examined post mortem. This was Heuck's[2] report on a family in which congenital ptosis affected the mother, aged 53; two sons, aged 18 and 16, and a daughter, aged 12. All had had complete ptosis since birth and showed defective ocular motility in

1. Wilbrand, H., and Saenger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1900, vol. 1, p. 85.

2. Heuck, G.: Ueber angeborenen vererbten Beweglichkeits-Defect der Augen, Klin. Monatsbl. f. Augenh. **17**:253, 1879.

all directions, especially in upward gaze. The 18 year old youth died, and both orbits were removed post mortem for examination. In the right orbit a poorly developed levator muscle, only 2 mm. wide, was found. In the left orbit no trace of the levator was detectable, even though special search was made for it. Examination of the other extraocular muscles showed that the superior rectus muscles were very thin and almost membranous and that the other rectus muscles were abnormally inserted into the globe. He made no study of the third nerve or of its nucleus. In 1895 Bach [3] reported a case of a girl aged 18 who had congenital ptosis, epicanthus and limitation of extraocular motility. After death large pieces of the muscles of the eye were removed. Microscopic study showed marked atrophy and poor development of the levator, as well as atrophy of the superior rectus. In 1895 Ahlström [4] described a case of complete congenital ptosis in which only a few tendon fibers were demonstrable in the right upper lid and there was no trace of a levator tendon in the left upper lid. Other anatomic defects of the extraocular muscles associated with congenital ptosis are [1] bifurcation of the internal rectus, failure of separation of the muscles, abnormal insertions, connective tissue strands in place of muscles and complete failure of the muscles.

A few authors have demonstrated changes in the third nerve, or in the nucleus of the third nerve, but in most of the cases ptosis has been associated with hydrocephalus or other disease of the central nervous system. At operation Bach [3] observed a case of congenital ptosis in which the levator tendon was demonstrated to be normally inserted and of normal length and width. He concluded, without examining the brain or the entire orbit, that here there must have been a defect of nerve conduction or an aplasia of the nuclear region of the third nerve. Siemerling [5] examined the brain and the orbits of a patient with mild unilateral congenital ptosis and inactive pupils. Autopsy showed leptomeningitis, a subdural hemorrhage and purulent bronchitis. Histologic examination of the brain and orbits showed mild degeneration of the levator muscle fibers on the side of the ptosis and bilateral degeneration of certain portions of the nucleus of the third nerve. He speculated that the cause of the ptosis in his case was a defective development of the nucleus of the third nerve. Wilbrand and Saenger [1] described degenerative changes in the nucleus of the third nerve of a man with bilateral congenital ptosis who died of tuberculosis at 47 years of age. They did not study the orbits or the levator muscles.

Several cases of congenital ptosis, associated with limitation of extraocular motility, are reported in the literature as instances of ophthalmoplegia externa. In 1924 Posey [6] reviewed the literature on anomalies of the extraocular muscles and added observations of his own. In 1927 Macklin [7] made some additional reports. Calhoun,[8] Stephenson,[9] Lawford,[10] Harman,[11] McCubbin,[12] de Schweinitz [13] and others [14] have contributed to this subject.

A review of the literature on the anatomic observations in cases of congenital ptosis shows that when such studies have been carried out some defect of the levator muscle has always been found. In those few cases in which defects have been noted in the third nerve or its nucleus, these defects could be explained on the basis of hydrocephalus, the presence of disease of the brain or an ascending degeneration of the third

3. Bach, L.: Anatomischer Befund eines doppelseitigen angeborenen Kryptophthalmos beim Kaninchen, Arch. f. Augenh. **32**:16-32, 1895-1896.

4. Ahlström, G.: Doppelseitige kongenitale Ptosis und Unbeweglichkeit der Bulbi, Beitr. z. Augenh. **11**: 523, 1895.

5. Siemerling, E.: Anatomischer Befund bei einseitiger congenitaler Ptosis, Arch. f. Psychiat. **23**:764-774, 1891-1892.

6. Posey, W. C.: Concerning Some Gross Structural Anomalies of the Muscles of the Eye and Its Adnexa, Arch. Ophth. **53**:344-354 (July) 1924.

7. Macklin, M. T.: Hereditary Abnormalities of the Eye, Canad. M. A. J. **16**:1340-1342 (Nov.) 1926; **17**: 55-60 (Jan.); 327 (March); 421 (April) 1927.

8. Calhoun, F. P.: Chronic Progressive Ophthalmoplegia Externa, South. M. J. **20**:923-925 (Dec.) 1927.

9. Stephenson, S.: A Case of Incomplete Congenital Ophthalmoplegia Externa, Ophthalmoscope **6**:678-685, 1908.

10. Lawford,, J. B.: Congenital and Hereditary Defects of Ocular Movements, Ophth. Rev. **6**:363, 1887; Congenital Hereditary Defects of Ocular Movements, Tr. Ophth. Soc. U. Kingdom **8**:262-274, 1888.

11. Harman, N. B.: Absence of Internal and External Recti Muscles, Tr. Ophth. Soc. U. Kingdom **25**:281-286, 1904-1905.

12. McCubbin, J. B.: A Case of Congenital Immotility, Extrinsic and Intrinsic, Am. J. Ophth. **28**:69-73, 1911.

13. de Schweinitz, G. E.: Complete Bilateral Congenital Ophthalmoplegia and Double Ptosis: Bilateral Cerebral Cortical Atrophy of Frontal and Parietal Regions (Encephalograms); Clinical Communication, Arch. Ophth. **5**: 15-28 (Jan.) 1931.

14. Steinheim: Blepharoptosis congenita und Defect der Muscul. recti superiores, Klin. Monatsbl. f. Augenh. **15**:99-102, 1877. Pflüger: Strabismus congenitus (2 Fälle), ibid. **14**:157, 1876. Natale, A.: Ptosis congénitas y anomalías musculares, Rev. de especialid. **3**:908-917 (Dec.) 1928.

nerve. Von Gudden,[15] Brouwer,[16] Chace[17] and others[18] have shown that removal of the eye or one of the extraocular muscles in very young animals results in degenerative changes in the nuclei concerned. Therefore, one would expect to find secondary changes in the nucleus of the third nerve in all cases of congenital ptosis. Also, the fact that the internal muscles of the eye are not affected in cases of ptosis is a strong point against the theory of the nervous origin of ptosis. Rodin and Barkan[19] were unable to find more than 1 undisputed case of hereditary congenital total ophthalmoplegia in the literature. Certainly, if congenital ptosis were due primarily to a lesion of the nucleus of the third nerve, the accommodation and pupillary reflexes should be affected more often. Furthermore, abnormal insertions, bifurcation and incomplete separation of the muscles and other anatomic defects associated with ptosis cannot be explained satisfactorily by the theory of a nervous origin.

Comparative Anatomy and Embryology of the Levator Palpebrae Muscle.—Comparative anatomy shows that the earliest extraocular muscles in the lowest vertebrates appeared simultaneously with the eye and have remained essentially unchanged throughout the entire vertebrate series. Indeed, except for the absence of the levator in the lower vertebrates, the number and nerve relationships of the extraocular muscles are essentially the same in man as in the dogfish.[20] The levator muscle appears first in the mammals and is the most recently acquired of the extraocular muscles.[21] The fact that recently acquired structures are more vulnerable to abnormal development may explain why the levator is often defectively developed in man.

Embryonically the levator muscle does not appear until comparatively late. The first trace of the extraocular muscles in man can be seen at the 7 mm. stage. During the 20 mm. stage the four rectus muscles and the two oblique muscles can be recognized.[22] The levator muscle develops later from the medial side of the superior rectus in the third month but does not reach its final position until the 75 mm. stage, or the fourth month. This pattern of development is of clinical interest because it helps to explain the cause of congenital ptosis. For example, if this developmental process should be hindered before the 20 mm. stage, any or all of the extraocular muscles will be defective. If development after the 20 mm. and before the 75 mm. stage should be hindered, the levator will be defective or absent. If the superior rectus fails to develop, the levator is always absent, although the reverse does not occur. This explains the frequent association of ptosis with weakness of the superior rectus and bears out the clinical observation that "no case of defective upward movement of the eye with perfect movement of the lid is known."[23]

The embryonic origin of Müller's[24] muscle of the upper lid is unknown. Ida Mann does not discuss this subject, nor do any of the standard textbooks on embryology. However, from comparative anatomy one learns that the anlage of Müller's muscle existed long before the levator appeared phylogenetically. Groyer[25] showed that in the water mammals this muscle is made up of striated muscle fibers but that in land mammals it consists of smooth muscle fibers. In the seal

15. von Gudden, cited by Howe, L.: The Muscles of the Eye, New York, G. P. Putnam's Sons, 1907, vol. I, p. 96.

16. Brouwer, B.: Klinisch-anatomische Untersuchung über den Oculomotoriuskern, Ztchr. f. d. ges. Neurol. u. Psychiat. **40**:152-189, 1918.

17. Chace, R. R.: Structural Changes in External Geniculate Body of Rat Following Removal of Eyes, Arch. Ophth. **30**:75-86 (July) 1943.

18. Hamburger, V.: The Effects of Wing Bud Extirpation on the Development of the Cerebral Nervous System in Chick Embryos, J. Exper. Zool. **68**:449-494 (Aug.) 1935. Bernheimer, S.: Die Reflexbahn der Pupillarreaction, Arch. f. Ophth. **47**:1, 1899; cited by Stevens, G. T.: Motor Apparatus of the Eyes, Philadelphia, F. A. Davis Company, 1906.

19. Rodin, F. H., and Barkan, H.: Hereditary Congenital Ptosis: Report of Pedigree and Review of Literature, Am. J. Ophth. **18**:213-225 (March) 1935.

20. Neal, H. V.: The History of the Eye Muscles, J. Morphol. **30**:433-453, 1918.

21. (*a*) Comparative Ophthalmology, in Wood, C. A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 4, p. 2621. (*b*) Walls, G. L.: The Vertebrate Eye and Its Adaptive Radiation, Bulletin 19, Cranbrook Institute of Science, 1942. (*c*) Waardenburg, P. J.: Die Zurückführung einer Reihe erblich-angeborener familiärer Augenmissbildungen auf eine Fixation normaler fetaler Verhältnisse, Arch. f. Ophth. **124**:221-299, 1930. (*d*) Collins, E. T.: Changes in the Visual Organs Correlated with the Adoption of Arboreal Life and with the Assumption of Erect Posture, Tr. Ophth. Soc. U. Kingdom **41**:10-90, 1922.

22. Mann, I.: The Development of the Human Eye, London, Cambridge University Press, 1928.

23. Mann, I.: Developmental Abnormalities of the Eye, London, Cambridge University Press, 1937.

24. Müller, A.: Ueber glatte Muskeln an den Augenlidern des Menschen und der Säugethiere, Verhandl. d. phys.-med. Gesellsch. in Würzburg **9**:244, 1859.

25. Groyer, F.: Vergleichende Anatomie des Musculus orbitalis und der Musculi palpebrales, Sitzungsb. d. k. Akad. d. Wissensch. Math.-naturw. Cl. **112**:51-100, 1903; Zur Anatomie des Musc. palpebral sup. des Menschen, Ztschr. f. Augenh. **14**:365, 1905; Ueber der Zusammenhang der Musculi tarsales mit den geraden Augenmuskeln beim Menschen und Säugetieren, Internat. Monatschr. f. Anat. u. Physiol. **23**:210-226, 1906.

and the dolphin, neither of which have a levator muscle, the superior rectus branches as it approaches the eyeball, one branch going to the globe and the other to the upper lid. In all land mammals Müller's muscle has become separated from the superior rectus and is a part of the levator. Groyer stated the belief that both the levator and Müller's muscle develop from the superior rectus but that Müller's muscle is older phylogenetically. This may explain why Müller's muscle has never been described as absent in cases of ptosis, probably because phylogenetically older organs are less vulnerable to atavism or to abnormal development than are newly acquired ones.

The treatment of congenital ptosis is primarily surgical and will be discussed with the various operations devised to remedy this condition.

HEREDITARY PTOSIS

Hereditary ptosis may manifest itself at birth (congenital type) or later in life (adult and juvenile types). The congenital variety is most likely due to a developmental defect of the levator muscle. Usually only one member of a family is affected, but sometimes congenital ptosis is familial and then can be transmitted either as a dominant or as a recessive characteristic. A number of excellent pedigrees on familial ptosis have been reported. In 1919 Briggs [26] presented a family tree in which hereditary congenital ptosis affected 64 members, distributed over 6 generations, both males and females being affected, according to the mendelian rule of dominance. In all but 1 of his cases the superior rectus was normal. Usher [27] published a pedigree in which bilateral ptosis associated with epicanthus affected 18 members, all the affected persons being in continuous line of descent from 1 affected male. The author pointed out that congenital ptosis without epicanthus is common, ptosis with epicanthus is less common and epicanthus without ptosis is rare. Ross [28] and McIlroy [29] also reported pedigrees showing association of ptosis with epicanthus. Rodin and Barkan [19] recorded bilateral ptosis with incomplete external ophthalmoplegia in a mother and 2 daughters. Rodin [30] later reported the pedigree of 4 generations in which hereditary congenital ptosis affected only one eye and the pedigree of 1 generation in which bilateral congenital ptosis was associated with epicanthus. Ptosis associated with blepharophimosis, affecting 21 out of 38 persons in 5 generations, was noted by Dimitry.[31] Bradburne [32] reported on a family in which bilateral ptosis associated with hereditary ophthalmoplegia affected 5 generations. De Schweinitz [13] described in detail the encephalograms of a patient with congenital ptosis and external ophthalmoplegia. Tatar and von Pap [33] reported cases of ptosis associated with mental weakness. Waardenburg [21c] describes defects of the inner canthus in patients with ptosis. A case of jaw-winking associated with ptosis was reported by Sinclair.[34] A rare type of paradoxic ptosis was described by Yanes.[35]

The noncongenital, or late developing, type of hereditary ptosis is rare. It was first described by Fuchs [36] in patients between 30 and 60 years of age. Collins [37] described a case of this type which he attributed to abiotrophy—a sort of hereditary lack of vital force affecting the levator muscle. Forsberg [38] cited the pedigrees of 67 persons, of whom 6 males and 6 females had the onset of ptosis between the ages of 5 and 20 years of age.

INCIDENCE OF PTOSIS

No figures are available for the incidence of ptosis in the general population, but hospital reports show that operations for ptosis comprise less than 1 per cent of all surgical procedures on the eye. Of 4,726 operations on the eye done

26. Briggs, H. H.: Hereditary Congenital Ptosis with Report of Sixty-Four Cases Conforming to the Mendelian Rule of Dominance, Am. J. Ophth. **2**:408-417 (June) 1919.

27. Usher, C. H.: A Pedigree of Epicanthus and Ptosis, Ann. Eugenics **1**:129-138, 1925.

28. Ross, N.: Congenital Epicanthus and Ptosis Transmitted Through Four Generations, Brit. M. J. **1**:378-379 (Feb. 27) 1932.

29. McIlroy, J. H.: Hereditary Ptosis with Epicanthus: Case with Pedigree Extending over Four Generations, Proc. Roy. Soc. Med. (Sect. Ophth.) **23**:17-20 (Jan.) 1930.

30. Rodin, F. H.: Pedigree of Four Generations of Hereditary Congenital Ptosis Affecting Only One Eye and Pedigree of One Generation of Congenital Ptosis with Epicanthus, Am. J. Ophth. **19**:597-599 (July) 1936.

31. Dimitry, T. J.: Hereditary Ptosis, Am. J. Ophth. **4**:655-658 (Sept.) 1921.

32. Bradburne, A. A.: Hereditary Ophthalmoplegia in Five Generations, Tr. Ophth. Soc. U. Kingdom **32**:142-153, 1911-1912.

33. Tatar, J., and von Pap, V.: Erbliche Ptosis und Schwachsinn, Arch. f. Ophth. **142**:627-633, 1941.

34. Sinclair, W. W.: Abnormal Associated Movements of the Eyelids, Ophth. Rev. **14**:307-319, 1895.

35. Yanes, T. R.: Paradoxic Monocular Ptosis, Arch. Ophth. **23**:1169-1172 (June) 1940.

36. Fuchs, E.: Ueber isolierte doppelseitige Ptosis, Arch. f. Ophth. **36** (pt. 1):234-259, 1890.

37. Collins, E. T.: Hereditary Ocular Degenerations—Ophthalmic Abiotrophies, Internat. Cong. Ophth. **1**: 103-143, 1922.

38. Forsberg, C. W.: Hereditary Ptosis, Journal-Lancet **52**:378-380 (June) 1932.

at Wills Hospital [39] in 1941, 32, or 0.69 per cent,[40] were for ptosis. The incidence of operations for ptosis ranked twenty-seventh in the list of operative procedures performed. Of 19,146 operations on the eye done at the New York Eye and Ear Infirmary [41] for the seven year period from 1936 to 1942 inclusive, 168, or 0.88 per cent, were for ptosis. The average number of operations for ptosis per year at this institution was 24. At the Manhattan Eye, Ear and Throat Hospital,[42] 2,971 operations on the eye were done in 1941, 17, or 0.57 per cent, of which were for ptosis.[40] In 1932 Moorfields Hospital, London,[43] had 3,276 "in patients," of whom 23, or 0.97 per cent, had operations, for ptosis. The ratio of congenital to acquired ptosis is about 3 to 1. In 1932 Moorfields Hospital did 23 operations for ptosis, 16 of which were for the congenital and 7 for the acquired type. In 1941 the New York Eye and Ear Infirmary did 26 operations for ptosis, 20 of which were for congenital and 6 for acquired ptosis.

The kind of operation for ptosis done at each hospital was generally not listed in the annual reports; but when the type was listed, it seemed to vary according to the locality of the hospital. At Moorfields Hospital, the Lexer operation (strips of fascia lata to the frontalis muscle) was generally done. Of the 17 cases in which operation was performed for ptosis at the Manhattan Eye, Ear and Throat Hospital, the Blaskovics procedure was done in 8, the Gillet de Grandmont operation in 3, the Eversbusch in 2, the Wheeler operation in 2, the Steatfield-Snellen operation in 1 and the Young operation in 1. At the latter institution, therefore, resection or shortening of the levator muscle was the procedure of choice.

CLINICAL ANOMALIES ASSOCIATED WITH CONGENITAL PTOSIS

A review of the literature on congenital ptosis shows that the following clinical anomalies may be associated with ptosis:

A. Abnormal extraocular motility
   1. Weakness of the superior rectus muscle
   2. Weakness of the superior rectus and the inferior oblique muscle
   3. Esotropia or exotropia
   4. Weakness or fibrosis of the external rectus muscle (retraction syndrome of Duane)
   5. More or less complete external ophthalmoplegia, with absence, bifurcation or abnormal insertion of the extraocular muscles

B. Abnormal development of the lids
   1. Epicanthus
      (*a*) Superciliary type—a fold of skin extending from the brow to the lower lid
      (*b*) Palpebral type—a fold of skin extending from the upper lid above the tarsus to the lower lid
      (*c*) Tarsal type—a fold of skin extending from the tarsal portion of the upper lid to the lower lid
   2. Blepharophimosis—an abnormally short fissure horizontally
   3. Abnormal inner canthus, e. g., an abnormally long inner canthal ligament, with defective development of the caruncle and the semilunar fold

C. Abnormal innervation of the levator muscle, producing jaw-winking (Marcus Gunn phenomenon)

D. Other abnormal developmental defects, such as extra toes and fingers and feeblemindedness

PREOPERATIVE STUDY OF PTOSIS

Before every operation to correct ptosis the following data should be determined:

1. The amount of ptosis in the eyes up, in the eyes straight ahead and in the eyes down position.

2. The amount of the function of the levator present, if any.

3. The extraocular motility of each eye, especially that of the superior rectus muscle.

4. The length of the palpebral fissures horizontally.

5. The position of the upper lid fold of each eye.

6. The vertical width of the tarsus.

7. The effect on the ptosis of instillation of drops of cocaine.

8. The vision of the two eyes and the presence of fusion, diplopia or amblyopia.

9. The sensibility of the cornea.

39. Report from the Wills Hospital, Philadelphia, 1941.

40. This percentage is low because minor operations, such as those for chalazion, were included in the statistics.

41. Annual Report of the New York Eye and Ear Infirmary, New York, 1936-1939; 1940-1942.

42. Report of the Manhattan Eye, Ear and Throat Hospital, New York, 1941.

43. Report of the Moorfields Hospital, London, 1932.

There is no entirely satisfactory way to measure ptosis. Dianoux and associates [44] used a perimeter, asking the patient to read a card as it was moved upward along the arc of the perimeter. The place where the lid covered the pupil and reading became impossible was noted and recorded in degrees as a measure of the ptosis present. This method has several disadvantages: It cannot be used with small children, with amblyopic eyes, or with paralysis of the superior rectus. Bulky equipment is necessary, and, what is more important, it is difficult to transpose the readings into figures which have meaning to the surgeon at the time of operation.

A second method consists in measuring the vertical width of the palpebral fissure of each eye in three directions of gaze, namely, with eyes looking up, with eyes looking straight ahead and with eyes looking down. This method has one disadvantage: namely, the movement of the upper lid is measured from a nonstationary object, the lower lid. It is well known that the lower lid moves upward when one is looking up and moves down when one is looking down. In cases of complete ptosis, in which there is no movement of the upper lid, the vertical width of the palpebral fissure may be 1 mm. or less with eyes looking up, 2 mm. with eyes straight ahead and as much as 5 or 6 mm. with eyes looking down. Measurements of this kind give an erroneous impression of the function of the levator because any variation in the width of the palpebral fissure is due solely to the rise and fall of the lower lid, and not to elevation of the upper lid. Furthermore, these measurements of ptosis are of little help to the surgeon at the time of operation in determining the amount of resection of the levator to be done because the base line, from which the measurements are taken, (the margin of the lower lid) is a nonstationary one.

A third method of measuring ptosis is to use the orbital rim at its highest point as the base from which to measure movements of the upper lid. This can be done with a compass, a millimeter rule, a Jameson rule or some similar instrument for measuring. While the patient is looking down, the examiner's thumb is placed above the patient's brow to steady one end of the rule, which is placed against or at the level of the superior orbital rim. The distance from the orbital rim to the margin of the upper lid is noted. The patient is then asked to look straight ahead, and the amount of elevation of the lid is noted in this position, as well as when he is looking up. This method measures directly the amount of ptosis and the elevation of the upper lid and, at the same time, prevents the frontalis muscle from affecting the measurements of ptosis. If the upper lid does not move upward when the eyes are looking up, complete ptosis is obviously present. With this method the surgeon can determine before operation the amount of correction needed and at the time of operation the amount of correction secured.

To the ophthalmic surgeon it is of the utmost importance to determine if possible the anatomic condition of the levator muscle before operation, especially if a resection of this muscle is contemplated. If the levator is entirely absent, it is utter folly to attempt resection of something which does not exist. The surgeon has at his disposal three methods of determining the functional state of the levator muscle. First, if the lid is elevated, the fissure widened or the upper lid fold deepened in upward gaze while the frontalis is kept from acting, it is proof of action of the levator. Second, if the superior rectus is weak, the levator is often poorly developed or weak. Third, if the fissure becomes wider after instillation of a few drops of cocaine, the levator is presumed to be present.[45]

44. Dianoux; Duverger, C., and Velter, E., cited by Sedan, J., in Bailliart, P.; Coutela, C.; Redslob, E., and Velter, E.: Traite d'ophthalmologie, Paris, Masson & Cie, 1939, vol. 7, p. 1066.

45. Ptosis due to paralysis of the sympathetic nerve supply to Müller's muscle (Horner's syndrome) may produce 3 to 4 mm. of ptosis and may be "cured" by the instillation of eye drops of cocaine (Lancaster, W. B.: Operative Treatment of Ptosis, Tr. Am. Acad. Ophth. **24**:235-263, 1919). This "cure" has been attributed to the action of cocaine directly on the smooth muscle of the upper lid. Certain authors have suggested the use of this test to determine the presence or absence of the levator muscle in cases of congenital ptosis. Adler (Clinical Physiology of the Eye, New York, The Macmillan Company, 1933, pp. 6-12) stated that cocaine has no effect in cases of congenital ptosis because the levator is absent and Müller's muscle has no fixed point from which to lift the lid. Briggs [26] reported that cocaine had no effect on the ptosis in his cases, and he assumed that this was due to the absence of the levator. Bradburne [32] reported on a pedigree in which congenital ptosis ran through 5 generations and affected 17 out of 37 members; in some cases there was no action of Müller's muscle, while in others "the action of Müller's muscle was plainly seen on attempting to open the eye." Apparently, the author did not use cocaine. Just how he determined that this muscle was present was not disclosed. Koster (Ztschr. f. Augenh. **3**:304-307, 1900; Nederl. tijdschr. v. geneesk. **35**:417-419, 1899; ab-

(Footnote continued on next page)

In addition to a study of the behavior of the levator, special attention should be paid to the function of the extraocular muscles. Associated with ptosis often is a weakness of elevation of the globe, which contraindicates any operation for ptosis until the hypotropia can be remedied. Especially is this true of those operations which utilize the lifting power of the superior rectus to support the ptosed lid. If the patient cannot roll his cornea upward under cover of the upper lid during sleep, exposure keratitis and loss of the eye may result. Also, exotropia and esotropia, if associated with ptosis, can best be remedied before the ptosis is corrected.

The horizontal length of the palpebral fissure should be measured in all cases of ptosis to rule out the presence of blepharophimosis. If this condition is present, the fissures should be lengthened before the ptosis is corrected.

The position of the upper lid fold of each eye should be recorded preoperatively, so that the surgeon will know at operation where to place the lid fold of the ptosed lid. Care in details like these produces better cosmetic results. The vertical width of the tarsus should be recorded preoperatively, so that the surgeon can estimate how much tarsus may be safely removed without producing a postoperative notch of the margin of the lid. De Blaskovics [46] advocated removing all but 1.5 mm. of the tarsus if necessary. Most authorities advocate leaving at least 3 mm. of tarsus in the midline to support the normal arch of the margin of the upper lid. The vertical width of the tarsus influences the amount of correction achieved in all operations which employ the superior rectus to elevate the lid (especially the operations of Trainor, Young, Greeves and Jameson), as well as the operation of tarsal resection, with or without resection of the levator.

Obviously, every patient with ptosis should have tests for visual acuity, fusion, diplopia and amblyopia. If the patient has good vision with fusion before operation, it is important to employ an operation for ptosis which is least likely to disturb his binocular vision. If diplopia is present before operation when the ptosed lid is elevated, this should be corrected before the ptosis is remedied. If amblyopia is present, the danger of disturbing fusion or of producing diplopia postoperatively is negligible, because here the object of the operation is primarily cosmetic. The tactile sensibility of the cornea should be noted preoperatively, for if the cornea is anesthetic one can be almost certain that uncovering the cornea will result sooner or later in neuroparalytic keratitis, with possible loss of the eye.

ANATOMY OF THE UPPER LID AND THE LEVATOR MUSCLE

From a special study of the orbits of cadavers and a careful perusal of the available textbooks [47] on the anatomy of the upper lid, one will note that the lid is made up of seven layers of tissue. These layers may be described, briefly, as follows:

1. The skin is firmly adherent to the underlying tissues of the brow and extends from the brow to the margin of the lid. About 5 or 6 mm. above the lashes the skin is thrown into a horizontal fold, called the upper lid fold, which marks the line of insertion of the terminal fibers of the levator aponeurosis. These fibers serve to keep the loose skin of the upper lid from prolapsing over the lashes. Elsewhere the skin is loosely attached to the underlying orbicularis muscle, except at the margin of the lid and at the internal and external canthal ligaments.

stracted, Jahresb. u. d. Leistung. u. Fortschr. im Geb. d. Ophth., 1899, p. 556) studied the effect on the upper lid of instilling drops of cocaine in the eyes of patients with congenital ptosis. He stated that if widening occurs it is proof that the levator is present. He assumed that Müller's muscle was always present in cases of congenital ptosis and concluded that if cocaine failed to elevate the lid the levator was absent. Mahoney and Sheehan (Experimental Ptosis in Primates, Arch. Neurol. & Psychiat. **35**:99-108 [Jan.] 1936) showed that after they had produced ptosis in monkeys by resection of the third nerve and the superior cervical ganglion, elevation of the lid occurred in fright or after the injection of epinephrine. They explained this elevation by assuming that the smooth muscle in the upper lid contracted even though the third nerve, the sympathetic fibers and the levator muscle were paralyzed. The value of the cocaine test for determining the presence of the levator muscle in cases of congenital ptosis seems at present somewhat uncertain, from available information.

46. de Blaskovics, L.: Treatment of Ptosis: Formation of a Fold in the Eyelid and Resection of the Levator and Tarsus, Arch. Ophth. **1**:672-680 (June) 1929.

47. (*a*) Whitnall, S. E.: Anatomy of the Human Orbit, ed. 2, New York, Oxford University Press, 1932. (*b*) Eisler, P.: Die Anatomie des menschlichen Auges, in Schieck, H., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, p. 198. (*c*) Hesser, C.: Der Bindegewebsapparat und die glatte Muskulatur der Orbita beim Menschen in normalen Zustande, Anat. Hefte **49**:1-302, 1913. (*d*) Kronfeld, P. C.; McHugh, G., and Polyak, S. L.: The Human Eye, Rochester, N. Y., Bausch & Lomb Press, 1943. (*e*) Wolff, E.: Anatomy of the Eye and Orbit, Philadelphia, P. Blakiston's Son & Co., 1936. (*f*) Hildreth, H. R.: Insertions of Levator Palpebrae Muscles, Am. J. Ophth. **24**:749-758 (July) 1941.

2. The orbicularis oculi muscle is fused with the corrugator supercilii muscle and the frontalis muscle at the brow. It extends downward from the brow to the margin of the lid, being loosely attached to the skin in front and to the septum orbitale and the tarsus behind. Medially it is attached to the nasal orbital margin and the internal canthal ligament and laterally to the bone of the temporal margin of the orbit and the external raphe. At the level of the upper lid fold the orbicularis is fused with some of the terminal fibers of the levator aponeurosis as these pass through the orbicularis to become attached to the skin.

3. The septum orbitale begins at the orbital margin from the junction of the periosteum of the frontal bone and the periorbita of the orbit. At this point it is thickest, and from here downward it becomes thinner, as it hugs the posterior surface of the orbicularis. As a matter of fact, it seems more accurate surgically to conceive of this structure as the posterior fascial sheath of the orbicularis than to think of it as a separate structure. At about the level of the upper border of the tarsus the septum orbitale fuses with the levator aponeurosis, thus forming a barrier to the prolapse of the orbital fat which is nearly always found at this junction. Anterior to the tarsus one finds a thin layer of loose connective tissue, which occupies the same relationship to the orbicularis muscle as does the septum orbitale above the tarsus and which, for practical purposes, can be considered a prolongation of this membrane. Medially the septum orbitale is adherent to the nasal orbital margin and laterally to the temporal orbital margin.

4. The orbital fat is posterior to the septum orbitale. It takes on a comma shape in sagittal sections, being thick and rounded in front and thin and pointed behind, and fills the triangular space between the sheath of the levator, below, the roof of the orbit, above, and the septum orbitale, in front.

5. Below and behind this roll of fat is the levator aponeurosis, which arises from the terminal fibers of the levator muscle and extends forward and downward to the tissues of the upper lid. It lies between the septum orbitale, in front, and Müller's muscle, behind, and is divisible, for description, into a medial and a lateral horn. The medial horn is thin and fragile and is difficult to follow as it passes from the levator muscle over the sheath of the reflected tendon of the superior oblique muscle to the upper border of the medial canthal ligament. The lateral horn is more dense and fibrous than the medial horn. It passes from the levator muscle to the upper border of the external canthal ligament and separates the lacrimal gland into two parts, so that the main part of the gland lies above the lateral horn and the accessory lobe behind it. The middle part of the levator tendon, flanked by the two horns, is the portion generally removed in resection of the levator muscle. It is about 10 to 12 mm. long and extends from the anterior end of the levator muscle toward the upper border of the tarsus, where, according to some authors, it seems to end. Careful dissection with a magnifying loupe will show that at the upper border of the tarsus the aponeurosis divides into thousands of fine fibrillae, which become attached partly to the anterior surface of the tarsus but chiefly to the overlying orbicularis and skin, producing the upper lid fold.

6. Müller's muscle is about 15 to 20 mm. wide and 10 or 12 mm. long and extends from the terminal fibers of the levator to the upper border of the tarsus. It is loosely adherent to the aponeurosis, on one side, and to the conjunctiva, on the other. Müller's muscle is about 0.5 to 1 mm. thick, is very delicate and is easily torn.

7. The conjunctiva is firmly adherent to the tarsus and loosely attached to Müller's muscle. The upper fornix is supported by the conjoined sheaths of the levator and the superior rectus muscles, i. e., by the so-called suspensory ligament of the upper fornix. Müller's muscle is so delicate and so thin that when the conjunctiva is reflected upward, the muscle is usually reflected also, unless minute care is taken to separate the two structures.

The levator muscle is about 50 mm. long, including its insertional attachments, and originates from the annulus of Zinn, at the apex of the orbit, directly above the origin of the superior rectus muscle. The under surface of the levator is in contact with the superior rectus all along its course. The fascial sheaths of these two muscles blend and become thicker as the globe is approached. This intimate association of the sheaths of the superior rectus and the levator may account for the occurrence of some elevation of the lid in upward rotation of the globe when the levator is completely paralyzed.

NERVE SUPPLY OF THE LEVATOR MUSCLE

As the third nerve enters the orbit through the superior orbital fissure and the annulus of Zinn, it branches into an upper and a lower division. The upper division supplies the superior rectus and the levator only. The inferior divi-

sion, besides supplying the internal rectus, the inferior rectus and the internal muscle of the eye, innervates the inferior oblique, which, together with the superior rectus, elevates the globe. The location of the higher centers, which control the synchronized up and down movements of the lid with the globe, have been definitely located in the vicinity of the angular gyrus of the frontal cortex.[48] The orbicularis, the antagonist of the levator, is supplied by the seventh cranial nerve. The higher centers controlling the reciprocal action of the orbicularis and the elevator must also be in the vicinity of the angular gyrus.

PHYSIOLOGY OF THE LEVATOR MUSCLE

The movements of the upper lid are partly voluntary and partly involuntary. The levator muscle, which lifts the upper lid, and the orbicularis, which closes the eye, are both synchronized physiologically with the elevators and the depressors of the globe. The associated movements of the lid and the globe are of four types:

1. Up and down movements. During the waking hours the action of the levator is automatically synchronized with the continuous up and down movements of the globe, while the orbicularis assumes a passive role.

2. Winking movements. Winking is an involuntary act, executed independently of the extraocular muscles, in which the action of the levator is synchronized with that of the orbicularis, so that the former relaxes while the latter contracts and vice versa.

3. Bell's phenomenon. During sleep the upper lid moves down and the globe moves up. Here the synchronous up and down movements of the lid and globe during the wakeful hours are broken, so that now the superior rectus contracts, the levator relaxes and the orbicularis contracts to close the lids.

4. The blink reflex. This is a sudden, firm closure of the eyelids, in which the superior rectus contracts to pull the globe up, the levator relaxes and the orbicularis contracts suddenly to close the lids. It may be initiated involuntarily by objects approaching the eyes suddenly, loud sounds or sudden flashes of light. Its physiologic basis is similar to Bell's phenomenon except that in blinking the orbicularis contracts more actively and completely.

Physiologically, Müller's muscle seems to be an auxiliary elevator of the upper lid. Anatomically, it serves as one of the attachments of the levator. Phylogenetically, it is a vestigial structure, which manifests itself functionally in man, both by overaction and by paralysis.[49]

SURGICAL TREATMENT OF PTOSIS

Undoubtedly, the incidence of ptosis has remained the same throughout the ages. Yet the ancients seemed to ignore the subject entirely in their writings. According to Valudi,[50] the only surgical procedure to survive from antiquity is the ancient Arabian operation of excising an oval piece of skin from the upper lid.

CLASSIFICATION OF OPERATIONS FOR PTOSIS

The eighty or more surgical procedures [51] designed for the cure of ptosis may be divided into three groups: [52]

1. Suspension of the upper lid from the brow, thus utilizing the action of the frontalis muscle for lifting the lid.

2. Attachment of the upper lid to the superior rectus, thereby replacing the action of the levator by the lifting power of the superior rectus.

3. Enhancement of the normal action of the levator by advancing, tucking or resecting this muscle.

48. Stevens, G. T.: Motor Apparatus of the Eyes, Philadelphia, F. A. Davis Company, 1906, p. 81. Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose and Throat, New York, Grune & Stratton, Inc., 1944. Rea, R. L.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938, p. 80.

49. Overaction of Müller's muscle of the upper lid occurs whenever the sympathetic nervous system is stimulated, as in fright or with hyperthyroidism or the acute phase of Horner's syndrome. This overaction is characterized by (1) a widening of the palpebral fissure, (2) apparent exophthalmos, (3) infrequent winking, (4) lid lag and (5) difficulty in everting the upper lid. After thyroidectomy in cases of hyperthyroidism, when overaction of the sympathetic nerve supply stops, the lid usually returns to its normal position, and the appearance of exophthalmos disappears. With paralysis of the cervical sympathetic fibers, Müller's muscle is relaxed, resulting in slight ptosis and apparent enophthalmos. Associated with the ptosis is narrowing of the pupil and diminution of sweating on the side of the lesion. Ptosis due to paralysis of Müller's muscle is slight and amounts to not more than 3 or 4 mm. and is "cured" temporarily by the instillation of cocaine eye drops, which causes contraction of Müller's muscle by acting directly on the smooth muscle fibers themselves.

50. Valude, cited by Wood.[104]

(Footnotes continued on next page)

Utilization of the Frontalis Muscle.—Dransart [53] was the first to conceive of producing cicatricial bands between the upper lid and the frontalis muscle. He used catgut sutures beneath the skin between the upper border of the tarsus and the brow. After Dransart's publication, a number of operations with modifications, based on this principle, were described. Pagenstecher [53] used silk sutures instead of catgut; Gayet [53] used platinum wire; Mules [53] used gold wire; Worth [53] used kangaroo tendon, and Harman [53] used gold chain. Other modifications were made by de Wecker,[53] Hess,[53] Landolt [53] and others.

These operations were a step forward in the surgical treatment of ptosis but on the whole were seldom entirely successful. All depended for their success on the development of cicatricial bands between the brow and the upper lid. In the days before antisepsis, infection usually occurred along the suture tracts, which produced firm bands of scar tissue. After antisepsis became a part of every operation, infection was rare and the operation more often a failure.

Not satisfied with the suture method of suspending the upper lid to the brow, surgeons began looking about for other means of attachment. Panus,[53] Allport,[53] Grimsdale,[53] Tansley [53] and others fashioned skin flaps from the upper lid and sutured them to the brow. Angelucci [53] and Sourdille [53] sutured the levator tendon (aponeurosis) to the brow, and Calderaro [53] tucked the tendon to the frontalis muscle. Machek [53] and, later, Reese [53] fashioned two flaps of the orbicularis and sutured them to the frontalis. Gifford [54] modified the Machek procedure to include both skin and the orbicularis in the flaps. Derby,[55] Wright,[56] Lexer,[57] Blair,[58] Weiner and Alvis,[59] Rosenburg,[60] Savin [61] and others used fascia lata. Fergus [53] and Roberts [53] used flaps of the frontalis muscle from the brow inserted into the upper lid.

The indication for the operation utilizing the frontalis muscle occurs primarily in those cases of ptosis in which shortening of the levator or suspension of the lid from the superior rectus cannot be done because of weakness of these muscles. Some authors consider the frontalis type of operation the method of choice in all cases of ptosis, while others mention it only to condemn it. The chief contraindication to its use occurs when the frontalis muscle is weak or paralyzed. Its chief advantage is that it can be used when the levator and/or the superior rectus are paralyzed. Its chief disadvantages are that the cosmetic result is not entirely satisfactory because the brow has to be elevated to raise the lid and because the pull is not in the right direction. Also, the flaps stretch; cysts form from buried skin; lagophthalmos sometimes results, and ugly puckering scars develop in the skin of the upper lid. Good results with this operation have been reported by Savin,[61] Blair and associates,[58] Poe,[62] Gifford,[54] Kiskadden,[63] Derby,[55] Rosenburg,[60] Magnus [64] and Butler.[65] Savin,[61] who performed the operation in over 50 cases, using strips of fascia lata anchored to the frontalis muscle, stated that it is the method of choice in all cases in which the levator muscle is weak and the frontalis muscle strong. Butler [65] reported on the results of 31 operations, using strips of fascia lata anchored to the frontalis muscle. Of these, a perfect result was obtained in 14, with normal closure and no notching of the margin of the lid; slight overcorrection resulted in 2; a puckering angulation of the upper lid was presented in 6, and slight undercorrection remained in 5.

51. De Blaskovics and Kreiker in their textbook on operations on the eye (Eingriffe am Auge, Stuttgart, Ferdinand Enke, 1938) list seventy-one surgical procedures for the treatment of ptosis. There are at least ten other procedures not mentioned by them.

52. An excellent classification of operations for ptosis is given by Wood.[104] The historical development of the surgical treatment of ptosis has been reviewed by Foster (Am. J. M. Sc. **118**:700-715, 1899), Beard [100] and others.

53. Cited by Wood.[104] Beard.[100] Elschnig, A.: Ptosisoperationen, in Handbuch der Augenheilkunde, Berlin, Julius Springer, 1922, vol. 1. Foster, M. L.: Am. J. M. Sc. **118**:700-715, 1899, Grimsdale, H., and Brewerton, E.: Textbook of Ophthalmic Operations, Baltimore, William Wood & Company, 1937, pp. 34-51. Spaeth.[105]

54. Gifford, S. R.: Machek Operation, Arch. Ophth. **8**:495-502 (Oct.) 1932.

55. Derby, G. S.: Correction of Ptosis by Fascia Lata Hammock, Am. J. Ophth. **11**:352-354 (May) 1928.

56. Wright, cited by Grimsdale, H., and Brewerton, E.: Textbook of Ophthalmic Operations, Baltimore, William Wood & Company, 1937, pp. 34-51.

57. Lexer, cited by Butler.[65]

58. Blair, V. P.; Brown, J. B., and Hamm, W. G.: Correction of Ptosis, Arch. Ophth. **7**:831-846 (Jan.) 1932.

59. Weiner, M., and Alvis, B.: Surgery of the Eye, Philadelphia, W. B. Saunders Company, pp. 323-346.

60. Rosenburg, S.: Fascia Lata Transplant for Ptosis, Am. J. Surg. **47**:142-148 (Jan.) 1940.

61. Savin, L. H.: Surgical Treatment of Ptosis, in Ridley, F., and Sorsby, A.: Modern Trends in Ophthalmology, London, Butterworth & Co., Ltd., 1940, pp. 573-581.

62. Poe, D. L.: Ophthalmoplegia Totalis, Am. J. Ophth. **16**:512-516 (June) 1933.

63. Kiskadden, W. S.: Surgical Treatment, Am. J. Surg. **27**:499-501 (March) 1935.

64. Magnus, J. A.: Correction of Ptosis by Two Strips of Fascia Lata, Brit. J. Ophth. **20**:460-464 (Aug.) 1936.

65. Butler, R. D. W.: Lexer Operation, Tr. Ophth. Soc. U. Kingdom **59**:579-585, 1939.

Of the many different surgical technics advocated for utilizing the lifting power of the frontalis muscle to elevate the upper lid, the use of strips of fascia lata seems to be the method of choice, all other methods having been generally discarded or being seldom used now. The chief reasons for failure of this method are slipping of the graft, infection and weakness of the frontalis muscle.

Utilization of the Superior Rectus Muscle.—In all operations utilizing the frontalis muscle the upper lid does not move synchronously with the globe. To meet this criticism, Motais [53] devised an ingenious operation in which he fashioned a flap of muscle tissue from the middle third of the superior rectus and sutured it to the upper lid. Parinaud [53] and Cannas [53] sutured the tendon of the levator to the superior rectus. These surgeons operated entirely through the conjunctiva. Shoemaker [66] and Taggart [67] modified the Motais operation by making an incision in the skin as well as in the conjunctiva. Later Kirby [68] did the operation entirely through the skin. Because the muscle flap sometimes pulled away after operation, Morgan [69] and Ellett [70] suggested removing a piece of sclera from the globe together with the muscle flap. Instead of using the middle third of the superior rectus, Stephenson [71] used the outer half of the muscle and Duverger and Velter [72] the inner half.

The small muscle flap sometimes slipped away after operation, resulting in failure; in other cases an exaggerated Gothic-like arch was produced. To avoid this and to produce a wider and stronger adhesion, Young [73] sutured the upper border of the tarsus directly to the tendon of the superior rectus. Greeves [74] fashioned two flaps from the upper border of the tarsus and sutured these to the medial and lateral borders of the tendon of the superior rectus. Trainor [75] formed a single flap from the superior border of the tarsus and, after passing it under the tendon of the superior rectus, sutured it to the upper border of the tarsus. Jameson [76] made a horizontal incision through the tarsus 2 to 3 mm. below its upper border and pulled the tendon of the superior rectus forward through this incision, so that the superior rectus became tucked between the tarsus and the skin. Dickey [77] placed a piece of fascia lata under the middle third of the superior rectus, while Weiner [59] and Gifford [78] passed it under the entire tendon of the superior rectus, each suturing the two ends of the fascia lata to the anterior surface of the tarsus. Not entirely satisfied with any of the aforementioned procedures, Wheeler [79] and Darier [80] fashioned two flaps of muscle tissue from the orbicularis in front of the tarsus and sutured them to the tendon of the superior rectus.

Most writers believe that the superior rectus operation is indicated only in those cases in which the superior rectus is intact and the levator is paralyzed. According to some authors, it should be used only when bilateral ptosis exists because of the danger of producing postoperative hypotropia and diplopia. If the cornea is anesthetic this operation is hazardous, because in sleep the lower part of the cornea is nearly always exposed, thus predisposing the eye to exposure keratitis. This complication is more likely if the elevators of the globe are weak. The chief advantage of this type of operation when it is successful is that it provides for the synchronous movement of the upper lid with the globe in looking up or down. A good lid fold develops, and good elevation of the lid is secured.

The disadvantages of this operation are as follows:

66. Shoemaker, W. T.: Observations on Motais Operation for Ptosis: Report of Three Cases, Ann. Ophth. **16**:608-615, 1907.

67. Taggart, H. J.: Modification of Motais Operation, Tr. Ophth. Soc. U. Kingdom **53**:417-421, 1933.

68. Kirby, D. B.: Modified Motais Operation for Blepharoptosis, Arch. Ophth. **57**:327-331, 1928; Blepharoptosis—The Technique of Its Surgical Correction, Surg., Gynec. & Obst. **70**:438-449 (Feb., no. 2A) 1940.

69. Morgan, O. G., cited by Greeves.[74]

70. Ellett, E. C., in discussion on Trainor.[75]

71. Stephenson, cited by Grimsdale, H., and Brewerton, E.: Textbook of Ophthalmic Operations, Baltimore, William Wood & Company, 1937, pp. 34-51.

72. Duverger, C., and Velter, E., in Bailliart, P.; Coutela, C.; Redslob, E., and Velter, E.: Traité d'ophthalmologie, Paris, Masson & Cie, 1939, vol. 7, p. 456.

73. Young, G.: An Operation for Congenital Ptosis, Brit. J. Ophth. **8**:272-275 (June) 1924.

74. Greeves, R. A.: Operation for Relief of Congenital Ptosis, Proc. Roy. Soc. Med. **26**:478-1482 (Sept.) 1933.

75. Trainor, M. E.: Operation for Lid Ptosis, Tr. Sect. Ophth., A. M. A., 1935, pp. 93-97.

76. Jameson, P. C.: Surgical Management with Special Reference to Use of Superior Rectus Muscle, Arch. Ophth. **18**:547-557 (Oct.) 1937.

77. Dickey, C. A.: Superior Rectus Fascia Lata Sling in Correction, Am. J. Ophth. **19**:660-664 (Aug.) 1936.

78. Gifford, S. R., and Puntenny, I.: Modification of Dickey Operation for Ptosis, Arch. Ophth. **28**:814-820 (Nov.) 1942.

79. Wheeler, J. M.: Correction of Ptosis by Attachment of Strips of Orbicularis Muscles to Superior Rectus Muscle, Tr. Sect. Ophth., A. M. A., 1938, pp. 130-137; Arch. Ophth. **21**:1-7 (Jan.) 1939.

80. Darier, cited by de Blaskovics.[46]

1. The upper lid cannot move independently of the globe, so that normal blinking [81] is interfered with.

2. Lagophthalmos is present during sleep, producing keratitis in 15 per cent of the cases.[54]

3. The operation is useful only when the superior rectus muscle is normal.

4. If the tongue of muscle slips, incomplete correction or complete failure will result.[46]

5. An exaggerated inverted V type of arching of the margin of the lid may result, especially with the classic Motais operation.[54]

6. Entropion, with in-turning of the lashes and abrasion of the corneal epithelium, may occur.[59]

7. Diplopia nearly always occurs during the first few weeks but usually disappears in three to four months.[72]

8. If fixation is maintained with the ptotic eye, the hypotropia will be exaggerated by overaction of the contralateral inferior oblique muscle.[81]

9. The Motais operation cannot be used with very young children because of the delicacy of the superior rectus.[82]

10. The operations done through the skin are considered difficult and produce considerable reaction.[79]

Good results with the Motais operation were reported by de Bassa,[83] Truc,[84] Lagrange [85] and Motais.[86] Since then, this type of operation has been advocated as the method of choice by Young,[73] Greeves,[74] Taggart,[67] Kirby,[68] Trainor,[75] Ellett,[70] Wheeler,[79] Shoemaker,[66] Jameson,[76] Dickey [77] and Cordes and Fritschi.[87] In 1928 Kirby [68] reported on 12 cases of operation for ptosis with his method. In 8 the operation was successful; in 2 cases the defect was undercorrected; in 1 it was overcorrected, and in 1 case the result was a failure. In 1936 Dickey [77] reported on 9 cases in which he had operated with his method; with failure in 1 case. In 1943 Cordes and Fritschi [87] reported on 21 other cases, with good results in all.

The chief reason for failure in all operations utilizing the superior rectus muscle is that the adhesions produced between the upper lid and the globe pull out or become stretched after operation. Of all the various methods advocated to produce this symblepharon, the use of fascia lata seems to be the most successful and the most popular at this time.

Resection of the Levator Palpebrae Muscle.—Bowman [88] was perhaps the first to perform this operation. He everted the upper lid, excised the upper edge of the tarsus with about ½ inch (1.27 cm.) of the levator tendon and sewed the conjunctiva and the levator tendon to the cut edge of the remaining tarsus. Bowman made no differentiation between Müller's muscle and the levator tendon, but in 1896 Wolff [89] called attention to this difference and described an operation for resection of Müller's muscle through the conjunctiva.[90] In 1909 de Blaskovics [91] first described his operation for resection of the levator muscle. His latest procedure [92] consists in everting the lid with a Desmarres lid hook, reflecting the conjunctiva upward, freeing the levator and its attachments (Müller's muscle and its aponeurosis) from the pretarsal tissues and excising 10 to 20 mm. of the levator muscle, together with 4 to 8 mm. of the upper part of the tarsus. In 1942 Agatston [93] modified the Blaskovics procedure slightly. For many years Wheeler [94] practiced, but never published, a modified Bowman resection of the levator muscle, in which a part of the tarsus and the attachments of the levator were removed through the conjunctiva. Maddox,[95] after reflecting the conjunctiva, cauterized Müller's muscle and the levator tendon to shorten the attachments of the levator muscle.

81. Dunnington, J. H.: Blepharoptosis—Its Surgical Treatment, M.Rec. & Ann. **35**:880-883 (June) 1941.

82. Bielschowsky, A.: Lectures on Motor Anomalies of the Eyes, Arch. Ophth. **13**:751-770 (May) 1935.

83. de Bassa, G., in discussion on Motais.[86]

84. Truc. P., in discussion on Motais.[86]

85. Lagrange, P., in discussion on Motais.[86]

86. Motais, M.: Quelques documents nouveaux sur l'operation de Motais, Paris, Angers, Germain and G. Grassin, 1907.

87. Cordes, F. C., and Fritschi, U.: Dickey Operation for Ptosis, Arch. Ophth. **31**:461-468 (June) 1944.

88. Bowman, W.: Roy. London Ophth. Hosp. Rep. **1**:34, 1859.

89. Wolff, H.: (*a*) Vorlägerung des Musculus levator palpebrae superioris mit Durchtrennung, Arch. f. Augenh. **33**:125, 1896; Ueber die Sehne des Musculus levator palpebrae superioris, Ztschr. f. Augenh. **13**: 440-457, 1905; **15**:576, 1906.

90. Wolff [89] also described an operation for resection of the levator tendon through the skin.

91. de Blaskovics, L.: Die Besciteung der Ptosis durch Verkurzung des hintern Lidsblattes und des Lidhebers, Klin. Monatsbl. f. Augenh. **47**:329, 1909.

92. de Blaskovics, L.: A New Operation for Ptosis with Shortening of the Levator and Tarsus, Arch. Ophth. **52**:563-573 (Nov.) 1923; footnote 46.

93. Agatston, S. S.: Resection of Levator Palpebrae Muscle by Conjunctival Route: Simplified Technique, Arch. Ophth. **27**:994-996 (May) 1942.

94. Wheeler, J. M., cited by Meek, R. E.: Applied Anatomy of the Eye: Relationship to Ophthalmic Surgery, Arch. Ophth. **26**:494-513 (Sept.) 1941.

95. Maddox, E. E.: A New Operation for Ptosis, Brit. J. Ophth. **1**:358-362 (June) 1917.

In 1896 Wolff [59] described a simple method for shortening the levator tendon through the skin, in which the levator aponeurosis was resected after being freed from the septum orbitale, anteriorly, and from Müller's muscle, posteriorly. Lapersonne [53] combined a resection of the levator tendon with an advancement, by suturing the cut edge of the levator tendon to the tarsus 2 or 3 mm. from the margin of the lid. Elschnig [53] resected the levator tendon in almost the same way except that he placed the incision 10 mm. above the convex border of the tarsus rather than 3 or 4 mm. below it. Lancaster [96] advocated shortening the levator through a cutaneous incision, removing a piece of skin and a piece of tarsus at the same time.

In 1883 Eversbusch [97] described an operation for enhancing the lifting power of the levator muscle by folding the tendon on itself. An incision was made through the skin, the orbicularis muscle and the septum orbitale, halfway between the brow and the margin of the lid, thus exposing the anterior surface of the levator tendon. The tendon was then tucked on itself by means of three mattress sutures on the anterior surface of the tarsus. Snellen [98] suggested two operations for tucking the levator, the first resembling the Eversbusch procedure and the other without a cutaneous incision. In the latter, he passed three sutures from the surface of the skin through the entire lid just above the tarsus; thence the needles were carried up to the fornix and then downward between the skin and the levator tendon, to emerge through the skin 2 mm. from the point of entrance. When the sutures were tied, the levator tendon was tucked on itself. Wilder [99] and Beard [100] have each described an operation for tucking the levator tendon and the septum orbitale by means of two sutures passed from the tarsus to the frontalis muscle, thus combining shortening of the levator with the frontalis type of operation.

Shortening of the Lid by Excision of a Part of It: The ancient Arabian procedure of excising a piece of skin from the upper lid was seldom successful [100] because the skin stretched so as to neutralize the effect of the operation. If enough skin was removed to get a permanent widening of the palpebral fissure, lagophthalmos, corneal ulceration and sometimes loss of the eye resulted. In 1863 von Graefe [101] modified the ancient Arabian procedure slightly by removing an elliptic piece of orbicularis instead of skin from the upper lid. Bowman,[102] apparently not satisfied with his first attempt to resect the levator through the conjunctiva, attempted two years later to shorten the levator through the skin. He incised the skin, reflected it upward and downward, excised an oval piece of orbicularis, tarsus and conjunctiva and sewed the edges together. Gillet de Grandmont [53] accomplished the same thing by placing the cutaneous incision 2 or 3 mm. above the margin of the lid instead of 8 mm. above, as was done by Bowman. Nicati,[53] Boucheron,[53] Gruening,[53] Theobald [53] and Mayou [53] resected a piece of the tarsus. Fergus [53] removed skin, orbicularis and tarsus but spared the conjunctiva. Galezowski [53] removed skin, orbicularis, tarsus and conjunctiva. With the possible exception of the Eversbusch operation, as advocated and modified by Meller,[103] none of these procedures done through the skin attained wide acceptance, and today practically all have been replaced by other methods.

Indications: Resection of the levator is indicated in all cases of ptosis in which this muscle is active. In all cases of complete congenital ptosis the levator is practically absent surgically, but in cases of acquired ptosis this muscle is present, even though paralyzed. In some cases the muscle can be shortened to a point where its tensile strength can be counted on to support the lid mechanically. Most authors (Wheeler,[79] Dunnington,[81] Kirby,[68] Wood,[104] Spaeth,[105] Weiner and Alvis [59] and others) agree that the levator muscle must be active, else resection of the muscle will give inadequate correction. However, de Blaskovics,[46] Lindner,[106] Jaensch [107] and others expressed disagreement with this opinion, maintaining that the Blaskovics procedure can be used for all types and degrees of ptosis with complete

96. Lancaster, W.: The Operative Treatment of Ptosis, Tr. Am. Acad. Ophth. **24**:235-263, 1919.

97. Eversbusch, O.: Zur Operation der congenitalen Blepharoptosis, Klin. Monatsbl. f. Augenh. **21**:100-107, 1883.

98. Snellen, H.: New Method of Treating Ptosis, Tr. Ophth. Soc. U. Kingdom **10**:207-210, 1890.

99. Wilder, W. H.: Operation for Ptosis, Tr. Am. Ophth. Soc. **8**:92-102, 1897-1899.

100. Beard, C. H.: Ophthalmic Surgery, Philadelphia, P. Blakiston's Son & Co., 1914, pp. 230-252.

101. von Graefe, A., cited by Beard.[100]

102. Bowman, W.: Roy. London Ophth. Hosp. Rec. **2**:111, 1861.

103. Meller, J.: Ophthalmic Surgery, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1923.

104. Wood, C. A.: System of Ophthalmic Operations, Chicago, Cleveland Press, 1911.

105. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery, ed. 3, Philadelphia, Lea & Febiger, 1944.

106. Lindner, K.: Ueber die Ptosisoperation nach Blaskovics, Klin. Monatsbl. f. Augenh. **93**:1-12 (July) 1934.

107. Jaensch, P. A.: Zur Ptosisoperation, Klin. Monatsbl. f. Augenh. **94**:183-189 (Feb.) 1935.

satisfaction, "even when the muscle is absent," because "an apparent function develops even in those cases in which this muscle is completely paralyzed or atrophic." [46]

Advantages and Disadvantages: According to Malbrán [108] and others, shortening the attachments of the levator muscle is the best procedure for the cure of ptosis and has the following advantages:

1. Postoperative reaction is negligible.

2. Lagophthalmos is almost absent.

3. The lid moves with the globe normally in all directions of gaze.

4. The margin of the lid is not deformed.

5. A good lid fold is produced.

6. The procedure can be used with slight or with severe ptosis.

7. The method can be used in cases of congenital ptosis even when accompanied with epicanthus.

8. Backward tilt of the head or elevation of the brow does not result.

9. There is no postoperative diplopia due to weakening of the superior rectus.

10. The method can be used in cases in which the superior rectus muscle is weak.

11. The winking reflex is not disturbed.

The disadvantages of the operation are as follows:

1. The operation can be used successfully only when the levator muscle is active.

2. There is a tendency to undercorrection, especially when insufficient muscle or tarsus has been removed.

3. Lagophthalmos may result if too much tissue is removed.

Reasons for Failure: Among the reasons advanced to explain why resection of the levator fails sometimes to give adequate correction are the following possibilities:

1. The levator may be paralyzed or too weak to support the lid. This may be true in some cases, especially when preoperative study reveals a weak levator muscle. As previously stated, however, de Blaskovics contended that the operation may be successful even though the levator is completely paralyzed.

2. The sutures may pull out after operation. This is especially likely to occur when inadequate sutures are used or when severe postoperative edema develops.

3. As Hildreth [47f] suggested, because of the failure of most authors to differentiate Müller's muscle from the levator muscle or its tendon during resection of the levator, only Müller's muscle, which is poor surgical tissue, may be resected, rather than the levator tendon, and this mistake may sometimes be the cause of inadequate correction. I [109] have shown by anatomic studies and operations on cadavers that it is almost impossible to resect Müller's muscle alone in any of the routine resections of the levator muscle through the conjunctiva.

4. Insufficient resection of the levator or its attachments may undoubtedly be the cause of inadequate correction in some cases. In a review of fifty-seven operations for shortening the levator muscle, I showed that only 1 or 2 mm. of correction as a general rule can be secured by the Bowman-Wheeler procedure. With the Blaskovics technic a larger amount of levator tissue can be removed, giving satisfactory correction in 91 per cent of the cases.[110] From this it was concluded that the Bowman-Wheeler operation, which is simpler to perform than the Blaskovics procedure, should be the operation of choice for slight degrees of ptosis and the Blaskovics technic for the larger amounts.

CRITERIA FOR THE PERFECT OPERATION FOR PTOSIS

According to Savin [61] and others, a successful operation for ptosis should meet the following requirements:

1. The curve of the margin of the upper lid should overlap the cornea equally on the two sides, covering the upper part of the cornea when the lids are normally open.

2. The margin of the lid should be a smooth curve and similar in the two eyes, with no tendency to form a notch or Gothic arch or to invert the lashes when the lid is widely open.

3. The superior palpebral crease should correspond on the two sides.

4. The eye should be capable of being widely opened and of shutting completely, both in forceful closure and in sleep.

5. Normal winking should be possible.

6. The vertical distance between the eyebrow and the margin of the upper lid should normally be the same in the two eyes.

108. Malbrán, J.: La corrección operatoria de la ptosis palpebral, Semana méd. **2**:1456-1462 (Dec. 18) 1941.

109. Berke, R. N.: Resection of the Levator Palpebrae Muscle for Ptosis. Arch. Ophth. **33**:269-280 (April) 1945; Tr. Am. Ophth. Soc., **42**:411-435, 1945.

110. de Blaskovics, footnotes 46 and 92. Lindner.[106] Jaensch.[107] Malbrán.[108]

7. The upper lid of each eye should move synchronously with the globe in all directions of gaze.

8. Diplopia or disturbing heterophoria should be absent.

It is doubtful whether any operation for ptosis ever completely achieved all of these requirements, especially in a case of unilateral complete congenital ptosis. Certainly, the frontalis type of operation does not give this degree of perfection, because the upper lid can be raised only by raising the brow. The superior rectus operation nearly always weakens this muscle, producing hypotropia, and leaves the lower part of the cornea exposed in sleep. Adequate resection of the levator muscle in cases of complete congenital ptosis often produces lagophthalmos in downward gaze. Therefore, in any case of complete congenital ptosis the surgeon must decide which of these criteria are the most important and which should be ignored.

To obtain a perfect cosmetic appearance and a complete functional result in a case of complete congenital ptosis, in agreement with the eight criteria listed here, must be a rare attainment. One is inclined to agree with Beard [100] when he stated: "All who have had much experience in this branch of ophthalmic surgery will agree that the results of ptosis operations, taken all in all, are far from brilliant."

SUMMARY

Pseudoptosis should be differentiated from true ptosis. The latter may be congenital or acquired, complete or incomplete, unilateral or bilateral. Acquired ptosis is usually due to a lesion of the nervous system and has many causes. Congenital ptosis is due to a lesion of the nucleus of the third nerve or to a developmental failure of the levator muscle. This form of ptosis usually occurs as an isolated case in an otherwise normal family, but it may be familial. Associated with congenital ptosis one may find weakness of the elevators of the globe, exotropia, esotropia, Duane's retraction syndrome or complete external ophthalmoplegia. Other associated congenital anomalies are epicanthus, blepharophimosis, abnormal inner canthus, jaw-winking, amblyopia and feeblemindedness. The preoperative study of ptosis should include measurement of the ptosis, determination of the function of the levator muscle present, effect of cocaine on the amount of ptosis present, study of the extraocular motility, measurement of the horizontal length of the palpebral fissure, notation of the position of the upper lid folds, measurement of the vertical width of the tarsus, determination of sensibility of the cornea and tests for visual acuity, fusion, diplopia and amblyopia.

The incidence of ptosis in the general population is not known. However, about 1 per cent of all major operations on the eye are for ptosis. The surgical treatment of ptosis can be divided into three categories: (1) utilization of the frontalis muscle to lift the upper lid; (2) substitution of the lifting power of the superior rectus muscle for that of the levator muscle; (3) enhancement of the lifting power of the levator muscle by shortening it. The success of the frontalis type of operation depends on the formation of cicatricial bands between the frontalis muscle and the upper lid. To create such bands, surgeons have used surgical gut, silk, kangaroo tendon, fascia lata, gold or platinum wire or gold chain. Other material consists of skin and/or orbicularis muscle from the upper lid or of the levator tendon. Some surgeons still consider this type of operation the procedure of choice, but most operators resort to this procedure only when the superior rectus or the levator palpebrae is too weak to support the lid. Its chief disadvantage lies in the puckering which occurs in the skin of the upper lid and the fact that the pull in lifting the upper lid is in the wrong direction.

The success of the operation utilizing the superior rectus muscle depends on the formation of cicatricial bands between the superior rectus and the upper lid. These may be formed by suturing a slip of the superior rectus to the tarsus or by attaching the tarsus or the levator tendon to the superior rectus. Other material used is strips of fascia lata or orbicularis from the lid, which are sutured to the superior rectus. The chief advantage secured by this operation is the excellent synchronization of movements of the upper lid with those of the globe in looking up and down. The chief disadvantage is lagophthalmos in sleep and the formation of a notch in the upper lid.

Resection of the levator muscle is the operation of choice when this muscle is not completely paralyzed. The levator may be shortened either by resection or by tucking a part of it through the conjunctiva or through the cutaneous routes. The chief advantage of this method is that it gives a natural appearance to the upper lid, with good elevation. The chief disadvantage is that it can be used only in cases of incomplete ptosis.

Anatomically, the upper lid is made up of seven layers of tissue, namely, skin, orbicularis, septum orbitale, orbital fat, levator aponeurosis, Müller's muscle and conjunctiva. Special attention should be paid to the levator aponeurosis and

Müller's muscle, for these structures must be identified in every operation of resection of the levator muscle. The physiologic movements of the upper lid may be divided into four groups, namely, (1) movements of the lid with the globe in looking up and down, (2) winking movements to moisten the cornea, (3) Bell's phenomena and (4) the blink reflex. Besides preserving each of these functions intact, every operation for ptosis should produce a good cosmetic appearance. Too much stress should not be placed on securing a good cosmetic result at the expense of normal function of the lid; else the cornea may suffer from exposure and ulceration.

CONCLUSION

Of the eighty odd surgical procedures advocated for the cure of ptosis, none is entirely satisfactory in all cases. As a general rule, resection of the levator palpebrae muscle is the operation of choice when this muscle is not paralyzed. Next in the order of preference is one of the operations designed to support the upper lid from the superior rectus muscle. If this muscle is weak or paretic, the frontalis type of operation may be utilized. In all operations for ptosis the functional result and the cosmetic appearance of the upper lid must be carefully considered.

430 Union Street.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

AMERICAN BOARD OF OPHTHALMOLOGY

Owing to transportation difficulties, the examination originally scheduled to be held in Los Angeles, Jan. 28 to 31, 1946, will be held in San Francisco, June 22 to 25, inclusive.

The following additional examinations will be held in 1946: Chicago, January 18 through 22; New York, April 10 through 13 (approximately); Chicago, October 9 through 12.

The officers for 1946 are: chairman, Edward C. Ellett, M.D., Memphis, Tenn.; vice chairman, Georgiana D. Theobald, M.D., Oak Park, Ill.; secretary-treasurer, S. Judd Beach, M.D., Portland, Maine; assistant secretary, Theodore L. Terry, M.D., Boston; consultant, Walter B. Lancaster, M.D., Boston.

Surgery List.—A new ruling requires that previously accepted candidates mail their surgery lists to the office of the Board at least sixty days prior to their examination. All new applicants are now required to send their list with application.

Ninth Annual W. Thornwall Davis Post Graduate Course in Ocular Pathology, Orthoptics and Surgery.—The ninth annual Wm. Thornwall Davis Post Graduate course in Ocular Pathology, Orthoptics and Surgery will be given at the George Washington University School of Medicine, Washington, D. C., Feb. 4 to 9, 1946. The course is limited to 30 registrants.

The course in pathology will be given by members of the staff of the Army Institute of Pathology: Colonel J. E. Ash, M.C., U.S.A., director of the Institute; Helenor Campbell Wilder; Captain Joyce Morris, M.C., A.U.S., and Lawrence Ambrogi.

The instructors for the course in surgery will be Dr. Ernest Sheppard, professor of ophthalmology; Dr. Ronald A. Cox, Dr. Richard W. Wilkinson, Dr. Sterling Bockoven, Dr. C. R. Naples, Dr. M. Noel Stow and Dr. J. Spencer Dryden.

Instructors for the course in orthoptics will be Dr. Frank D. Costenbader, Mary Everist Kramer, Louisa Wells, Dorothy R. Bair, Alice L. McPhail and Marjorie Enos.

Bequest to College of Physicians of Philadelphia.—By the will of the late Dr. Paul L. Sartain, of Philadelphia, the sum of $5,000 was bequeathed to the College of Physicians of Philadelphia, in trust for the creation of a "Library of Ophthalmological Illustrations." The trustee is to acquire by purchase or through gifts to the college, drawings and paintings delineating faithfully and accurately, as well as artistically, the ocular structures. High class reproductions of illustrations in the ophthalmologic field, as made by photographic or other methods, may be accepted, and photographs, lantern slides and films of any kind for projection on the screen may be selected. All acceptable specimens are to be properly mounted and catalogued and so arranged as to be readily consulted and used by the fellows of the College and other accredited visitors. No efforts should be spared for the careful handling and preservation of such a library.

In addition, Dr. Sartain provided that "if at any time there should be a surplus of income from the fund, such surplus may be used for the preparation of illustrations of interesting cases submitted by those unable to bear the expense of their delineation, but such illustrations shall become the property of the college and be included in the Sartain library."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

SEROLOGIC STUDIES IN ACUTE EYE DISEASES. C. C. GRAY and W. A. MOOR, Am. J. Ophth. **28:** 180 (Feb.) 1945.

Gray and Moor give the following summary:

"1. The sera of 16 eye patients were used in the manner of the Neufeld test to determine the presence of an agency that would produce a nonspecific capsular swelling of Type XXVII pneumococcus.

"2. Two cases showed a positive reaction.

"3. The etiology of these two cases was determined to be an organism in the gram-negative or the gram-positive groups.

"4. After removal of the cause of the choroiditis, the test became negative." W. S. REESE.

ANTERIOR MEGALOPHTHALMOS AND ARACHNODACTYLY. W. V. STEPHENSON, Am. J. Ophth. **28:** 315 (March) 1945.

Stephenson reports a case of arachnodactyly with anterior megalophthalmos. Emphasis is given to the genetic aspects of the disease. There are many variations of anterior megalophthalmos, owing to the infinite disturbances in the genes. The term anterior dysophthalmos is suggested for the condition. W. ZENTMAYER.

Congenital Anomalies

OCULAR MALDEVELOPMENT IN EXTREMELY PREMATURE INFANTS. RETROLENTAL FIBROPLASIA: VI. GENERAL CONSIDERATION. T. L. TERRY, J. A. M. A. **128:** 582 (June 23) 1945.

Over 10 per cent of the infants born very prematurely, with a weight of 3 pounds (1,306 Gm.) or less at birth, can be expected to be blind because of retrolental fibroplasia. If this incidence continues, at least 600 cases will occur annually, thus increasing materially the number of the blind.

The typical characteristics of retrolental fibroplasia are an opaque, vascularized membrane behind the lens; microphthalmos; shallow anterior chamber; fetal blue color of the iris; thin ciliary processes in front of the opaque tissue; searching nystagmus; apparent photophobia; persistent hyaloid artery, and, often, retinal detachment.

The basis of the disease lies in the presence of a functioning hyaloid artery–tunica vasculosa lentis system in all infants born even three or four weeks prematurely.

Of all the probable causes listed, precocious exposure to light is considered the most tenable, and preventive measures should be taken.

For prevention, mydriatics and miotics should be used, but if these are considered too drastic, the infants should be kept in a dark room with a red light and the eyes covered adequately during examinations and nursing care.

Treatment consists in preventing the most common complications, glaucoma and posterior synechia, by use of miotics and mydriatics. Surgical therapy and irradiation have proved unsuccessful. However, a surgical attempt to establish a new vascularization of the ciliary body with the episclera is being made, with some beneficial results. W. ZENTMAYER.

TOTAL ALBINISM OF THE EYES: REPORT OF A CASE. O. CORREA NETTO, Arq. brasil. de oftal. **7:** 13 (Feb.) 1944.

Netto reports the case of a man 22 years of age with total depigmentation of the eyeballs. The patient has a brother and a maternal uncle who are albinos. The rest of the family, including several brothers of the patient, are normal. The function of the retina of albinos is not involved. Dark adaptation and perception of colors are normal. Avitaminosis A and B play no role in albinism. The condition is familial, hereditary, recessive and dominant and is favored by consanguinity. Marriage of albino persons should be interdicted for eugenic reasons. W. ZENTMAYER.

Conjunctiva

AMYLOID DISEASE OF THE CONJUNCTIVA. N. B. ELLES, Am. J. Ophth. **28:** 486 (May) 1945.

Elles reports a case of amyloid disease of the conjunctiva and concludes that amyloidosis may manifest itself in the conjunctiva as a primary, and perhaps as a secondary, disease.

The fact that in most cases reported the disease has followed or complicated trachoma need not mean that trachoma is a factor in its development, for in the majority of cases of trachoma amyloidosis does not occur. The geographic location of most cases of ocular involvement, that is, the Baltic states, Russia, Japan and China, where deficiency diseases are more prevalent, would indicate that deficiency may be a factor. Biochemically, the process of amyloid deposit appears to be a disturbance in endogenous protein metabolism, in which the rate of formation of catabolic products exceeds the ability of the tissues to dispose of them. Histologically, the

evidence points to primary involvement of the phagocytic reticuloendothelial cells, whose location is exactly in the places where amyloid is constantly seen. It might be desirable to abandon the term "degeneration" and to refer to the condition as amyloid disease. W. ZENTMAYER.

SELF-INFLICTED CONJUNCTIVITIS: AN ACCOUNT OF CASES PRODUCED BY THE JEQUIRITY AND CASTOR OIL SEEDS. E. J. SOMERSET, Brit. J. Ophth. **29:** 196 (April) 1945.

Of 31 patients seen, the condition was unilateral in 19 (right eye only in 8 and left eye only in 11) and bilateral in 12. All were Indians, none of whom had gained the rank of commissioned officers. They came from widely separated units. It was thought that in the majority of cases the condition was due to the use of the castor oil or the jequirity seed. Typically, the patient keeps the eye closed and frequently has a guilty and sulky appearance. Three main clinical types are seen: subacute, hyperacute and chronic.

The author describes a personal experiment to show that the inner seed of the castor oil plant is capable of producing conjunctivitis. The castor oil plant and the jequirity plant are described, and the clinical signs, pathologic diagnosis, differential diagnosis and treatment are discussed.

The article is illustrated. W. ZENTMAYER.

Cornea and Sclera

RECURRENT EROSION OF THE CORNEA. P. A. CHANDLER, Am. J. Ophth. **28:** 355 (April) 1945.

Chandler recognizes two types of recurrent erosion of the cornea: the macroform and the microform. He suggests use of the ophthalmoscope with a + 4.00 D. lens to detect the lesion and believes that damaged endothelium may cause the persistent epithelial edema. He recommends the use of boric acid ointment (10 per cent) at night for the microform type and scraping the cornea and chemical cauterization of the denuded area for the macroform type.

W. S. REESE.

EPIDEMIC KERATO-CONJUNCTIVITIS IN THE MIDDLE EAST: CLINICAL AND EXPERIMENTAL STUDY. A. FEIGENBAUM, I. C. MICHAELSON and W. KORNBLÜTH, Brit. J. Ophth. **29:** 389 (Aug.) 1945.

Three hundred and eighty-one cases of keratoconjunctivitis were investigated in the Middle East, chiefly in Palestine. The majority of these cases occurred during epidemics. In the Middle East the beginning of the rainy season appears to be the period of maximum incidence of the disease. In Palestine the disease, although now occurring inland, first manifested itself in the coastal plain. This is in accordance with observations in other parts of the globe.

In some cases trauma appeared to play a definite role in determining the onset of the condition. The involvement of lids, conjunctiva, regional glands and cornea formed a well defined clinical picture, differentiable for the most part on clinical grounds from swimming pool conjunctivitis, vernal conjunctivitis, trachoma, influenzal conjunctivitis and herpes cornea. In 60.4 per cent of cases the cornea was involved. In the cases without corneal lesions the diagnosis was made on the clinical picture, the absence of bacteria, typical smears and the occurrence during epidemics.

The mildest forms of therapy proved the best. Douchings with cool water, instillation of epinephrine hydrochloride (1:1,000) and, if necessary, use of cocaine or atropine drops were the most satisfactory remedies. Energetic treatment prolonged the course of the disease and caused gross changes in the conjunctiva. Histologic examination of the human conjunctiva showed flattening of the epithelium, pronounced capillary dilatation and edema in the subepithelial tissue, subepithelial infiltration with lymphocytes and large mononuclear cells, multiple mitoses and, in cases of the chronic type, considerable hypertrophy of the papillae.

Histologic examination of a rabbit's cornea inoculated five days previously showed the typical lesions of macular keratitis. Experiments with rabbits confirmed the specific infective nature of the causal agent. This agent was not infective for mice with different methods of inoculation.

Tissue culture and filtration experiments showed that the infective agent is a filtrable virus. Clinical immunity was apparently present in the blood of convalescent patients.

Epidemic keratoconjunctivitis is a specific disease of the conjunctiva and cornea, caused in all probability by a filter-passing virus, which can produce a similar condition in the cornea of the rabbit. The disease has its own well defined epidemiologic and clinical features. Treatment must not be energetic. Douching with cool water and instillation of epinephrine and, if necessary, cocaine and atropine drops are the most helpful remedies. W. ZENTMAYER.

BILATERAL MARGINAL DYSTROPHY OF THE CORNEA. J. F. BALZA and F. K. CRAMER, Arch. de oftal. de Buenos Aires **19:** 73 (Feb.) 1944.

A case of bilateral marginal dystrophy of the cornea (keratoectasia) is described, in which spontaneous rupture of Descemet's membrane, with subsequent improvement in vision, occurred in the right eye. Prior to the accident the upper segment of the cornea was prominent and extremely opaque. After rupture of Descemet's

membrane, the cornea was flattened and became almost transparent. Visual acuity improved from 1/10 to 10/10, with a correction of —2.00 D. sph. ⊃ —2.00 D. cyl., axis 90.

The authors point out that the good results of the accidental rupture in this case suggest that a discission of Descemet's membrane might be accomplished purposely in the treatment of this condition.

The article is illustrated, and a bibliography is furnished. H. F. CARRASQUILLO.

BLUE SCLEROTICS ASSOCIATED WITH BONY DEFECTS IN THE NOSE. E. CHAN, Chinese M. J. **63:** 55 (Jan.) 1945.

A Chinese girl aged 18 years, in addition to blue sclerotics, had a cystic tumor which occupied the entire nasal fossa, apparently invading the ethmoid and sphenoid sinuses. There were pressure changes in the surrounding bony structures. The left eye was proptosed and was later enucleated. The sclera was from one-third to two-thirds the normal thickness but was of unequal tenuity. There was a slight decrease in the number, but no change in the size, of the fibers. There was thinning of the cornea.

W. ZENTMAYER.

SCLERAL ABSCESS. F. M. LEE, Chinese M. J. **63:** 60 (Jan.) 1945.

Lee reports 2 cases of scleral abscess. The first case was that of a boy aged 8 years. After an iridectomy the patient tore off the surgical dressings and rubbed the eye. The abscess developed one week later. In the second case, that of a young man, the abscess developed one month after an iridectomy. W. ZENTMAYER.

TUBERCULOMA OF THE SCLERA. K. S. SUN, Chinese M. J. **63:** 67 (Jan.) 1945.

Tuberculoma of the sclera was noted in a young Chinese woman. The growth was excised and subjected to histologic examination. Clinical studies indicate that it was secondary to a tuberculous process in the lung. A brief review of some of the reported cases is incorporated. The mode of infection and the question of diagnosis are briefly discussed. W. ZENTMAYER.

KERATOCONUS: REPORT OF A CASE. W. S. MAO, Chinese M. J. **63:** 90 (Jan.) 1945.

Keratoconus was observed in the right eye of a man aged 37. Both eyes were affected with trachoma stage III. The right eye also had concomitant convergent strabismus — vision was limited to perception of light. The case is of interest because of the rarity of the keratoconus in the western and northern part of China.

W. ZENTMAYER.

General

A DISCUSSION OF OCULAR MALINGERING IN THE ARMED SERVICES. A. C. UNSWORTH, Am. J. Ophth. **28:** 148 (Feb.) 1945.

Unsworth discusses malingering in the armed services, its types and the various tests for its detection. He recognizes several forms of malingering, which may be classified as positive, in which the man attempts to prove his falsification, and negative, in which he falsely denies the existence of a history or physical signs of existent disease or injury.

There is a significant relationship of malingering, war neurosis and morale among the troops. High morale depends on several local factors in the soldier's environment: healthy training conditions; food of adequate amount and good quality; sufficient exercise; supervised sports and other diversions; medical care that inspires confidence; good relationship between the men and their officers; rigid discipline and drilling, which inspires a sense of group security; talks to groups of soldiers on the psychologic aspect of fear and its understanding, and, most important, a clear perspective and appreciation of the ideology for which the war is being fought and the value to the individual, his family, the nation and the world of maintaining this ideology.

W. ZENTMAYER.

OPHTHALMOLOGY IN PARIS DURING THE WAR. P. PRELAT, Am. J. Ophth. **28:** 1073, (Oct.) 1945.

Prelat gives an interesting account of ophthalmologic work in Paris during World War II. He presents numerous brief reports of cases and of theses submitted to the Ophthalmological Society of Paris, the last bulletin of which carried reports from October to December 1939.

W. S. REESE.

THE EYE OF THE WEST AFRICAN NEGRO. J. G. SCOTT, Brit. J. Ophth. **29:** 12 (Jan.) 1945.

A study of the eyes of 1,000 Gambian school children and 1,100 Gambian, 300 Gold Coast, 300 Nigerian and 300 Cameroon soldiers, in addition to hospital patients and outpatients seen at a West African military hospital over a period of eighteen months, was made. Noteworthy differences were found between the eyes of Africans and those of Europeans.

A summary follows:

Pigment of the interpalpebral conjunctiva and a ring of pigment around the limbus are normal.

Vascularity of the cornea beyond the ring of pigment is pathologic and is most frequently caused by trachoma. This condition should be excluded before pannus is attributed to tropical disease.

The Negro fundus is red, not chocolate or slate gray, in appearance.

A 2 per cent solution of homatropine hydrobromide and/or cocaine hydrochloride is not an efficient mydriatic.

Nebulas in the substantia propria are common and are most frequently caused by onchocerciasis and trachoma.

Iritis is caused by onchocerciasis and by trypanosomiasis, as well as by more usual diseases.

Microfilarias are not uncommon in the aqueous, where they are well tolerated. The most common, if not the only one, is Onchocerca volvulus.

The value of removal or destruction of nodules containing the adult Onchocerca volvulus is doubted, as the nonencapsulated worm plays an important part in infection and in producing ocular complications. W. ZENTMAYER.

EYE DISEASES IN THE EAST. J. MINTON, Brit. J. Ophth. **29:** 19 (Jan.) 1945.

Minton gives a brief account of the ophthalmic diseases of the natives of Iraq. Epidemics of infective ophthalmias occur regularly every year. They begin in May and last until December. About 60 per cent of the population is thought to be infected with trachoma. The British troops showed a very low incidence of infective conditions of the eye. There was no epidemic of conjunctivitis. The Indian troops had a much higher incidence of conjunctivitis than the British. Mucopurulent conjunctivitis of a severe type associated with corneal ulceration was of frequent occurrence. The Indian and Ceylonese troops and the natives suffered from epidemic punctate keratitis, but no cases were seen among the British troops. There were no cases of gonococcic conjunctivitis among the British or the Indian troops. W. ZENTMAYER.

General Diseases

ARIBOFLAVINOSIS. I. MANN, Am. J. Ophth. **28:** 243 (March) 1945.

Mann reports a case of ariboflavinosis in a man aged 29 years. The right eye showed severe conjunctival injection, the main vessels being intensely engorged. The limbal plexus was full. There were no new conjunctival vessels. The cornea showed marginal vascularization and opacities in the substantia propria. The vascularization was distinctive. It was in the form of parallel, radially arranged loops springing from the apexes of the loops of the limbal plexus and extending in three arcades beyond this slightly more than halfway to the margin of the pupil. The vessels were so engorged with blood that they appeared to stand out from the surface. They were mostly subepithelial, but in the region of the opacities in the cornea they had penetrated the substantia propria to about one-half its thickness. They extended all around the circumference of the cornea. The opacities were coarse, whitish patches, located in the subepithelium. Both eyes were affected, but to a different degree.

The patient had been on a diet which contained no fruit and supplied green vegetables once a week.

With a proper diet and the administration of multivitamins, the cure was dramatic.

W. ZENTMAYER.

HERPETIC DISEASES OF THE EYE IN THE LIGHT OF MODERN VIRUS INVESTIGATIONS. M. GRUTTER, Arch. Soc. de oftal. hispano-am. **3:** 494 (Nov.-Dec.) 1943.

The herpes virus may be the causal agent in the following processes: herpes of the palpebral margin, vesicular keratitis in its varied forms, dendritic keratitis in its variations, geographic keratitis, stellate keratitis, filamentous keratitis, recurrent corneal erosions, striate keratitis, circumscribed parenchymatous keratitis, diffuse parenchymatous keratitis, herpetic endothelial keratitis and herpes of the irises. Epidemic keratitis and nummular keratitis may have some connection with the virus. An explanation of how these conditions are brought about by the action of the virus toxin is given.

H. F. CARRASQUILLO.

Hygiene, Sociology, Education and History

PATRON SAINTS OF THE EYES: AN OUTLINE. F. L. P. KOCH, Am. J. Ophth. **28:** 161 (Feb.) 1945.

Koch recounts the histories of the various saints and the ocular conditions which they were thought to influence. He stresses the importance of sympathetic understanding of faith in such beliefs. W. S. REESE.

A CENTRAL OPHTHALMOLOGICAL INSTITUTE FOR THE UNITED NATIONS. A. LOEWENSTEIN, Brit. J. Ophth. **29:** 6 (Jan.) 1945.

Loewenstein suggests a common central institute of ophthalmology for the United Nations. Similar institutes for other specialties might follow. They may be distributed over the large towns of the Nations according to the need and the availability of suitable teachers. In the near future the common budget of the Allied Nations will be considerable. The idea of a common education must not be forgotten. Central medical institutes are a part of it, and all nations will enjoy the fruits of their work. A blueprint of such an institution is offered, and the details of equipment are given. W. ZENTMAYER.

WORD BLINDNESS; ITS CAUSE AND CURE. R. HALL, Brit. J. Ophth. **29:** 467 (Sept.) 1945.

The author suffered from word blindness, being unable to read up to the age of 15, i. e., prior to puberty. (Did altered thyroid function have something to do with this change?) The theory has been advanced by Stout that the chief instrument used by the blind in perceiving the shape and size of objects is the hand, or, rather, the two hands. This is accomplished by what may be called synthetic touch, because it yields a total simultaneous impression of all, or many, parts of the object. Second, the finger tips may explore the parts and contours of the object by gradually moving over them. This may be called the active touch, or the analytic touch, because it analyzes or breaks up into a series of successive impressions what synthetic touch presents as a simultaneous whole. This concept naturally leads to the question whether development in definiteness of the perception of letter order, or words, may not be essentially due to the intimate union and cooperation of synthetic and analytic vision. All words must be analyzed or broken down into their smaller parts and then synthesized again by association. When the letters are thoroughly known, two letter words should be learned. These should be learned first of all by analytic vision, that is, by splitting them up into, or regarding them as, two separate letters, until sufficient synthetic vision is acquired to fuse them by association or to see them as a single unit. Passages in books should then be used, the patient picking out all known two letter groups, such as those underlined in "*N*ow th*is is the be*st *me*thod." This is a form of analytic vision which, by breaking down words, will gradually extend synthetic vision so that it will gradually absorb even the longer words thus broken down. For "now" is no longer a meaningless collection of letters—it is the word "no" with a letter after it—that is, in its written form it has acquired a definite personality which differentiates it from other words; and all thought has to move by way of differentiation.

When all two letter words have been thoroughly mastered in this way, the same procedure should be followed with three letter words, and then with four letter words, in order gradually to extend synthetic vision. W. ZENTMAYER.

THE ONE-EYED WORKER. J. MINTON, Brit. J. Ophth. **29:** 472 (Sept.) 1945.

Minton divides one-eyed people into two groups: those who have been without an eye since childhood and those who have lost the vision of an eye in adult life as the result of disease or injury. Persons of the first group are in no way handicapped and have full confidence in the carrying out of their work. A person of the second group takes a little time to adjust himself to the new visual condition. This adjustment requires from a few weeks to several months. The degree of depth perception acquired depends on several factors: intelligence and previous experience in judging distance, age, sex and whether the eye lost was the dominant eye. The time required for the regaining of confidence to return to work should rarely exceed two to three months, but the greater number of men stay away from work for periods varying from six to nine, or even ten months. The reasons for this are fear and lack of confidence, compensation and litigation difficulties and the unwillingness of firms to employ one-eyed workers. The occupation suitable for a one-eyed person depends on the vision of the good eye. With vision of 6/6 or 6/9, all occupations involving close work can be performed with comfort. If visual acuity is 6/12, occupations requiring constant close work are inadvisable. If vision is less than 6/12, such work as gardening, farming or domestic service is suitable. W. ZENTMAYER.

Injuries

REPORT OF AN EYE INJURED BY LIGHTNING. L. B. SHEPPARD, Am. J. Ophth. **28:** 195 (Feb.) 1945.

A youth aged 18 years was struck by lightning and remained unconscious over four hours. On the right side there were marked edema of the lids, moderate chemosis of the conjunctiva and complete external ophthalmoplegia. The cornea showed a whitish gray opacity, with cloudiness in the posterior half of the cornea. Vision was limited to light perception, and tension was normal. There was retinal edema about the optic papilla. On the twenty-ninth day there were localized detachment of the retina, two small areas of choroiditis in an area of retinal edema and a fibrous band in the posterior part of the vitreous. On the forty-fifth day a second area of detachment of the retina was noted. About one week after the accident an acute attack of iridocyclitis developed. Two months later there were opacities in the posterior cortex and the vitreous and complete detachment of the inferior portion of the retina. W. ZENTMAYER.

INTRAOCULAR FOREIGN BODY OF INFREQUENT LOCALIZATION AND PROLONGED TOLERANCE. J. M. VILA ORTIZ, Arch. de oftal. de Buenos Aires **19:** 91 (Feb.) 1944.

Vila Ortiz describes a case in which a foreign body was lodged in the papilla. According to him, this is the third case of the kind reported in the literature. A man 36 years of age fell

and received an injury to the left eye. The eye showed hyphemia and turbidity of the media, but no portal of entry of any foreign body was found. The roentgenographic examination also failed to show its presence. The giant magnet was also used for diagnosis, with negative results. After the media cleared, a grayish mass 2 by 2.5 mm., taken to be a foreign body, was observed in the papilla. The eye was blind. A second roentgenogram, taken with the Sweet method, corroborated the diagnosis. The only sign of intolerance was optic neuritis. No endeavor was made to remove the foreign body. Four years later the eye was quiet. Foreign bodies in this location are well tolerated and do not require extraction, nor is enucleation indicated if the foreign body is chemically inert and nonmagnetic. The article is illustrated.

H. F. CARRASQUILLO.

Instruments

AN OPHTHALMIC SLIDE RULE. TIENYUNG MIAO, Chinese M. J. **63:** 80 (Jan.) 1945.

On each side of the rule there are several graduations from which may be determined the interpupillary distance, the near point of convergence, the binocular parallactic angle, the angle of convergence, the accommodative power, the tangent rule, the meter angles, etc.

The article is illustrated.

W. ZENTMAYER.

Lacrimal Apparatus

DACRYORHINOSTOMY. N. BELMONTE GONZALES, Arch. Soc. oftal. hispano-am. **4:** 33 (Jan.-Feb.) 1944.

When the lacrimal sac has been removed and the canaliculi are intact, the operation of Arruga, canaliculorhinostomy, is indicated.

When there is absence of the lacrimal sac and the canaliculi, the operation of dacryorhinostomy is indicated. This procedure is also used when the obstruction is in the canaliculi before they empty into the sac.

The operation which the author terms dacryorhinostomy, and which consists in opening a fistulous passage between the lacus lacrimalis and the nasal cavity, is described in detail, and the procedure is illustrated with colored plates.

H. F. CARRASQUILLO.

Lens

TRANSPARENCY OF THE LENS FOLLOWING TRAUMATIC CATARACT. H. J. STERN, Brit. J. Ophth. **29:** 48 (Jan.) 1945.

The left eye of a Negro aged 25 had been injured by a flying particle of stone. Several hours after the injury an anterior capsule cataract was present. Exactly the same abnormality was seen in the other eye. Three days later a brilliantly white, mushroom-shaped mass was protruding from the lens into the anterior chamber of the left eye. The roentgenogram was noncontributory. Thirteen days after the injury, only a few gray capsular opacities were left, and a feathery posterior cortical cataract was seen. On the twenty-third day the eye was again white and perfectly normal except for the unchanged anterior capsular cataract and a fine opacity near the center of the cornea. Vision was 6/6. The eye was emmetropic. The author analyzes the various possibilities in explanation of the case.

W. ZENTMAYER.

Lids

THE ORIGIN AND PURPOSE OF BLINKING. A. HALL, Brit. J. Ophth. **29:** 445 (Sept.) 1945.

This inquiry was begun some years ago in order to find out to what extent the rate of blinking is reduced in chronic encephalitis. In the course of preliminary tests on normal persons certain unexpected points were noted. All persons tested were unaware that their blinking was being watched; in each test the surroundings and the procedure were the same for all subjects; all stimuli capable of causing local reflex blinking were excluded.

Apart from the reflex blinking seen in animals and man, which is solely for the local protection and efficient action of the eyes themselves, all blinking in man is the muscular act by which the controlling nervous center automatically shuts off the act of fixation in order to put the extramuscular reflex into full action.

Reasons for blinking may be as follows:

For self preservation, either from a threat of real danger to life coming from without through any of the sense organs or from undefined fears aroused by the surrounding conditions at the time. The former is the primitive unconditioned reflex seen throughout the animal kingdom; the latter is probably of the same origin but is conditioned in the primates by the increased security of arboreal life and in man still more, by the requirements of civilization. This type forms a large proportion of the blinking in everyday life.

For change in the direction of fixation. While some blinking is but a part of the orientation of the head and eyes in the self preservation reflex, it far more often, especially in the primates, serves either for the purpose of "looking about" or as an aid to the various purposes for which fixation is used independently.

For pauses in the act of reading aloud. These are conditioned reflexes acquired by training.

W. ZENTMAYER.

Neurology

HEAD INJURIES FROM THE OPHTHALMOLOGIST'S VIEWPOINT. A. D. ECKER and E. W. ANTHONY, Brit. J. Ophth. **29:** 43 (Jan.) 1945.

The article discusses some of the newer concepts concerned with head injuries which may be of interest to the consulting ophthalmologist.

Concerning the late results of blunt head injuries, the vast majority of both civilian and military personnel return to their full duties in a matter of weeks, with proper management. The patient must be approached in an attitude of calm reassurance.

The fixed dilated pupil is an infallible sign of increased intracranial pressure. It is usually due to a laterally placed intracranial lesion on the same side, which is most often in the temporal, but may be in the frontal or the parietal, lobe. The occurrence of bilateral fixed dilated pupils soon after a head injury indicates a bad but not hopeless prognosis. If the condition is associated with neurogenic hyperthermia, rapid pulse, quickly rising rectal temperature, cold skin and decerebrate rigidity, there is damage to the midbrain.

The Argyll Robertson pupil may result from a head injury. The lesion in cases in which this sign is present may be either in the central nervous system itself or in the peripheral efferent pathway to the pupil. In a study of lesions of the temporal lobe, one can use routine clinical, roentgenologic and electroencephalographic methods, as well as observation of the visual fields, speech and caloric nystagmus.

When recovery from a muscular palsy occurs within a matter of days, the nerve has been injured in a process akin to concussion, whereas if months are required, the axons presumably have been torn and have regenerated in an intact nerve sheath. If no recovery occurs, one can assume severance of the nerve, severe damage to its blood supply or such firm scarring that regeneration is prevented. Changes in the visual fields are discussed. The presence of normal visual fields does not exclude the possibility of a slowly progressive subdural hematoma overlying the optic radiations.

It has been shown that electroencephalograms, when used as an index of active cerebral abnormality after head injury, will help to estimate the extent of the cerebral damage.

W. ZENTMAYER.

Ocular Muscles

ORTHOPTIC FICTIONS AND MISCONCEPTIONS. L. WELLS, Am. J. Ophth. **28:** 890 (Aug.) 1945.

Wells discusses various misconceptions and differences of opinion relating to orthoptics. One misconception is that vision in an eye with amblyopia ex anopsia cannot be improved after the sixth year of life. Such a conception is an admission that occlusion has been incomplete and of too short duration. Another fiction in orthoptics is that "alternating squints cannot be made to fuse." It is a misconception to assume that anomalous correspondence cannot exist in a patient who has visual acuity of 20/15 in each eye.

W. ZENTMAYER.

SOME OBSERVATIONS ON A TENDENCY TO NEAR-POINT ESOPHORIA AND POSSIBLE CONTRIBUTORY FACTORS. D. D. STENHOUSE STEWART, Brit. J. Ophth. **29:** 37 (Jan.) 1945.

A short period of exacting close work in special conditions of binocular dissociation is shown to cause a change of a temporary nature in ocular muscle balance at the near point (33 cm.) as assessed by normal routine methods.

It is implied that muscle balance at the near point cannot accurately be stated without reference to the manner of use of the eyes preceding the assessment.

If it can be assumed that man is deficient in natural power of voluntary divergence to counteract accommodative esophoria, it follows that in the "resting" state "near point" exophoria of some degree accompanied with adequate reserve power of voluntary convergence is desirable for the comfortable performance of sustained close work.

It is further suggested that in some cases temporary disturbance of this balance and of comfortable near point effort can arise from emotional states, especially if aroused by close work and related to anxiety about its successful performance.

W. ZENTMAYER.

OPERATION FOR CORRECTION OF PARALYTIC EXTERNAL (LATERAL) RECTUS PALSY. J. P. SPENCER WALKER, Brit. J. Ophth. **29:** 477 (Sept.) 1945.

Walker reports 2 cases of paralyses of the external rectus muscle in which a slight modification of Lancaster's operation was used. In the first case, that of a child 4 years of age, the history suggested birth injury to the right eye. The eye was amblyopic. On the twenty-fifth day after the operation the eye could be abducted fully. In the second case, that of a child 6 years of age, the left external rectus muscle had been palsied since birth. There was some postural torticollis. Immediately after operation there was an overcorrection; at the end of two weeks the power of adduction was returning and the torticollis was greatly improved. The modification of the Lancaster operation consisted in the recession of the internal rectus muscle.

W. ZENTMAYER.

STRABISMUS. BARBOSA DA LUZ, Rev. brasil. de oftal. **2:** 31 (March) 1944.

Barbosa da Luz discusses the respective indications for orthoptic and surgical therapy of strabismus. He discusses the value of determination of visual acuity, angles of deviation during ocular movements, type of strabismus, sensory condition of the retina, presence and quality of binocular vision and Worth's test of four points for the selection of either type of therapy. The author concludes that operation followed by orthoptic training is the proper therapy for strabismus. Surgical therapy alone is indicated only for patients who ask only for cosmetic correction of strabismus. W. ZENTMAYER.

Operations

A MODIFICATION AND EXTENSION OF THE McREYNOLDS OPERATION FOR PTERYGIUM. L. STATZ, Brit. J. Ophth. **29:** 193 (April) 1945.

The first step of the operation is the same as that in the McReynolds method. The new direction of the displaced pterygium is such that there is no overlapping of the conjunctiva along the upper portion of the limbus. The redundant edge of the conjunctiva, between the incision and the tied silk stitch, is incised and a flap is formed; this flap is turned upward and stitched to cover the raw area of sclera and to lie along the limbus without encroaching on the cornea. A stitch from the adjacent conjunctiva biting firmly into the flap serves to obliterate a small uncovered area. The accompanying sketches show the steps of the operation. W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

BULGING OF EYELIDS WITH EXOPHTHALMOS. F. F. RUNDLE and C. W. WILSON, Clin. Sc. **5:** 31 (Aug. 7) 1944.

In this paper, Rundle and Wilson direct attention to the fatty swelling of the lids occurring with exophthalmos in cases of diffuse toxic goiter, giving particular attention to the clinical characteristics and the mechanism. Such swelling is not confined to patients with toxic diffuse goiter but is found with a variety of conditions characterized by overfilling of the orbit and exophthalmos. It is, in fact, simply a protrusion of the normal lid and is not due to edema of the palpebral tissues, as is generally stated. Variations in protrusion or recession accurately reflect changes in the degree of orbital filling. In wasting of the orbital tissues the eye is sunken. The lids are receded. Bulging of the lids due to prolapsing fat has a well defined clinical appearance. In the upper lid this is most evident with the eye closed; in the lower lid, with the eye open. After discussing prolapsing orbital fat as the cause of bulging of the lids, the authors show how it is differentiated from edema of the eyelids. Further, they demonstrate the correlation between protrusion of the lid and proptosis and then discuss the mechanics of exophthalmos and protrusion of the lid. Disparity between the bulk of the contents and the capacity of the orbit may arise from a variety of clinical conditions, but some expression, such as orbital overfilling, overcrowding or disproportion, is necessary to describe their common pathologic basis. In its strict sense, exophthalmos is only one of the consequent signs. Protrusion of the lid is another. It remains for clinical investigation to determine others and so to decide which of the manifold ocular signs described in cases of toxic diffuse goiter are explicable on this simple basis. Unfortunately, "exophthalmos" is often used in an omnibus sense to describe all such cognate signs. The authors conclude that variations in the plane of the eyelids, whether they are sunken, normal or bulging, reflect corresponding variations in the degree of orbital filling, as do correlated changes in the position of the eye. The clinical characteristics of sunken and bulging lids are well defined; these, together with the exophthalmometric reading, make possible the detection of departures from the average degree of orbital filling.

J. A. M. A. (W. ZENTMAYER).

UNILATERAL HYPOTONY DURING ANAESTHESIA. INA BRITTAIN and G. J. C. BRITTAIN, Brit. M. J. **1:** 442 (March 31) 1945.

At the Derbyshire Royal Infirmary unilateral hypotony was observed after anesthesia in 13 of a series of 300 cases. The cause was pressure exerted by the rim of a badly adjusted face mask. According to the authors, pentothal sodium is known to cause bilateral reduction in tension and tends to exaggerate the effects produced by pressure. The possibility of the occurrence of acute glaucoma in the affected eye should be borne in mind, as the lowered intraocular tension is followed by a rise above the normal, which may last for some hours. ARNOLD KNAPP.

Physiology

DYNAMIC FACTORS IN THE FORMATION AND REABSORPTION OF AQUEOUS HUMOUR. J. S. FRIEDENWALD, Brit. J. Ophth. **28:** 503 (Oct.) 1944.

During the last decade the problem of the aqueous has been attacked by three groups of workers, using three different technics. Duke-Elder and his co-workers have studied the composition of the aqueous, have found the fluid to be hypertonic in electrolytes and have consequently concluded that secretory forces enter into its formation. Kinsey, Grant and Cogan

have studied the rate at which dissolved substances are exchanged between blood and aqueous, have found that this rate is greater for electrolytes than for nonelectrolytes (excluding water) and have concluded that electrolytes are secreted into the intraocular cavity, whereas diffusion and osmosis suffice to account for the rates of exchange of nonelectrolytes provided reasonable assumptions are made regarding the mechanism of outflow. Friedenwald and his co-workers have studied the mechanism of secretion in the ciliary body and have been able to formulate with some precision how the metabolic energies of this tissue are utilized for the work of secretion.

The present paper represents an effort to bring together these three independent lines of investigation. It is shown that the secretory mechanism as it is at present understood through the work of Friedenwald and his co-workers would account for the formation of an aqueous hypertonic in electrolytes, as has been found by Duke-Elder and his co-workers, and would likewise account for the higher rate of transport of electrolytes than of nonelectrolytes from blood to aqueous, as has been found by Kinsey and his colleagues. The position of the three groups of workers may be summarized as follows: Duke-Elder has concluded that the aqueous is a secretion. Kinsey agrees and specifies that it is the electrolytes in particular that are secreted. Friedenwald agrees and specifies that of the electrolytes it is primarily the cations that are secreted. W. ZENTMAYER.

Retina and Optic Nerve

COMPLETE CONGENITAL PIGMENTATION OF THE OPTIC DISC. W. MOEHLE, Am. J. Ophth. **28:** 377 (April) 1945.

Moehle briefly reviews the literature and reports the case of a 65 year old man with chronic glaucoma, whose right optic disk was entirely covered with brownish black pigment.

W. S. REESE.

SHEATHING OF THE RETINAL VEINS IN MULTIPLE SCLEROSIS. C. W. RUCKER, J. A. M. A. **127:** 970 (April 14) 1945.

In a series of 50 cases of sheathing of the retinal veins, multiple sclerosis was present in 31; it was suspected in 11 cases, and there was no evidence of the disease in 8 cases. Thickening of the vascular walls at a distance from the disk is almost always indicative of disease. Venous sheathing that occurs without any other visible disease in the retina seems to have special significance. The fundal picture bears resemblance to a feature observed by a number of pathologists in sections of the central nervous system of patients who have died of multiple sclerosis, namely, an accumulation of cells around venules. Rucker concludes that sheathing of some retinal veins is occasionally encountered on ophthalmoscopic examination of otherwise normal fundi. It is usually indicative of disease of the central nervous system, most often multiple sclerosis. The article is illustrated. W. ZENTMAYER.

STARGARDT'S DISEASE: REPORT OF THREE CASES. E. SALGADO BENAVIDES, Arch. Soc. oftal. hispano-am. **4:** 185 (March-April) 1944.

After observing 3 patients with heredomacular degeneration, the author comes to the following conclusions: 1. Stargardt's disease is to be considered a retinal degeneration, beginning at the macula, familial in nature and of unknown cause. 2. Its characteristic progressive course is divided into three stages. In the first stage subjective manifestations appear in relation to retinal lesions. At this time the lesions are visible only on careful observation. The second stage coincides with well defined retinal lesions limited to the macular area. The third stage is characterized by pigmented and degenerated foci in extensive areas of the retina. 3. The disease seems to have a selective tendency to involvement of the retinal cones. 4. The process does not produce total blindness.

H. F. CARRASQUILLO.

ETIOPATHOGENESIS OF "ESSENTIAL" ARTERIAL HYPERTENSION: PRESENT CONCEPTION OF PROBLEM. A. VILELA, Rev. brasil. de oftal. **2:** 181 (June) 1944.

According to Vilela, the main pathogenic factor in essential arterial hypertension is worry, emotion and subconscious conflicts. Heredity and the presence of renal and endocrine disorders are associated factors. The stage of the disease and the prognosis for the patient's life are determined by the progressive changes in the fundus of the eye as evaluated by Wagener, who places the patient according to the changes in the fundus in one of four groups.

In patients of the first and second groups the disease is in its early stages and consequently is benign. It is in the progressive, intermediate, stage in patients of the third group and is malignant in patients of the fourth group, with a prognosis of one or two years of life in 95 per cent of the cases. W. ZENTMAYER.

Trachoma

TRACHOMA VIRUS AND THE MORPHOLOGY OF INCLUSION BODIES. A. LOEWENSTEIN, Am. J. Ophth. **28:** 282 (March) 1945.

Loewenstein summarizes his observations on advanced trachoma: The follicles consisted of lymphocytes, plasma cells, fibroblasts and macrophages (cystophages, Marchand). The macro-

phages contained no granules, suggesting inclusion bodies. The caps reported by Cuenod in the cells of the follicles were not found in any of the 23 cases of advanced trachoma which formed the basis of this histologic investigation. The appearance of the inclusion bodies differed in scrapings and in section. In the scrapings they showed considerable polymorphism, and individual elementary and initial bodies might be found alone in the cells. In sections encapsulated inclusion bodies with many Giemsa staining elements were found in the superficial epithelial layers. In the middle layers they were fewer, and they were absent in the deeper layers. In the subepithelial tissues, reticulum cells of fibroblast character were filled with blue and purple granules. Granules were free in the tissues — some single, others in colonies. The tarsus was found to be full of reticulum cells of fibroblast character, similar to those in the subepithelial tissues. W. ZENTMAYER.

THE ETIOLOGY OF TRACHOMA: A CRITICAL REVIEW OF PRESENT KNOWLEDGE. J. O. W. BLAND, Brit. J. Ophth. **29:** 407 (Aug.) 1945.

The available evidence shows that trachoma is an infectious disease, caused by a noncultivable, filtrable agent identical with the elementary and initial bodies found in the Prowazek-Halberstaedter inclusions and having a close relation to the viruses of inclusion blennorrhea, lymphogranuloma venereum and psittacosis. There is no evidence that this agent possesses an arthropod host or that the louse is a vector of the disease, though the agent may survive for some days in the body of the louse. In what group of organisms this agent, with the other three to which it is related, is to be placed becomes largely a matter of personal choice. All four agents resemble the rickettsias in a number of points. They differ from the majority of rickettsias in not possessing an arthropod host. The four agents differ from the typical large virus in possessing initial bodies which stain blue with the Giemsa method and in showing greater pleomorphism, in forming inclusions with a basophilic matrix and in staining with Castaneda's stain. They differ from the typical rickettsias in the lack of an arthropod host, in not showing bacillary and thread forms and in forming inclusions with a matrix. The author considers that they stand in an intermediary position between the rickettsias and the large viruses and may possibly form a biologic link between them. He prefers to group them with the viruses but to give them a distinctive position as the "basophilic viruses" on account of the blue staining of their initial bodies and of the matrix of their inclusions, a reaction which distinguishes them from the larger typical viruses, which do not possess blue initial bodies and whose inclusions are acidophilic.

A bibliography is given. W. ZENTMAYER.

QUININE BISULFATE IN LOCAL THERAPY OF TRACHOMA. O. APRIGLIANO, Arq. brasil. de oftal. **7:** 45 (April) 1944.

Aprigliano observed the results of three types of therapy in three groups, of 60, 30 and 30 patients (children and adults), with trachoma. The therapy for patients in the first and second groups consisted in the expression of the conjunctival granulations with a swab soaked in a 7.5 per cent solution of quinine bisulfate and massage for two minutes, followed by the local applicataion of a second and a third swab soaked in the same solution. The caruncle was massaged separately. A 1 per cent solution of tetracaine hydrochloride was used for conjunctival anesthesia. Local quinine bisulfate therapy was employed every other day for two weeks and twice a week thereafter until a cure was effected. An ointment which contained 6 per cent quinine bisulfate was applied locally on the days in which the local therapy was not applied. In the second group, only sulfanilamide by mouth, in proper doses, was administered. The patients in the third group had the usual therapy for trachoma which is given in dispensaries, namely, digital expression of the granulations and conjunctival massages with solutions of acid mercuric nitrate and of copper sulfate. The results of quinine therapy were the same for patients who had sulfanilamide as for those who did not. The duration of local therapy with quinine bisulfate, whether used alone or in association with sulfanilamide, was greatly shortened as compared with that for patients who had treatment with acid mercuric nitrate and copper sulfate. Quinine bisulfate, locally applied to eyes with trachoma, exerts a prompt favorable effect on the corneal lesion, the pannus and the keratitic ulcers. The treatment is well tolerated. It is advisable to give short courses of quinine bisulfate at long intervals after cure, in order to prevent recurrences. W. ZENTMAYER.

A STATISTICAL STUDY OF TRACHOMA AMONG IN-PATIENTS. HSIU-HSIANG CHI, Chinese M. J. **63:** 73 (Jan.) 1945.

Of 2,903 patients admitted to the department of ophthalmology of the Chengtu Eye, Ear, Nose and Throat Hospital from January 1940 to December 1943, 1,305 (44.9 per cent) had a condition diagnosed as trachoma. The sex ratio was 54.6 per cent females to 45.4 per cent male. The age of 65.1 per cent of the patients ranged from 16 to 40 years. More than half the patients were affected during the most useful part of their lives. Housewives had the highest incidence, followed by students, merchants and farmers. Nearly all affected persons showed sequelae or complications; 31.9 per cent were "blind" on admission, while 22.8 per cent were so classified on discharge. In 80 per cent the cause of blindness was a lesion of the cornea due directly or indi-

rectly to trachoma. In order to reduce blindness in China, it is essential therefore to control the spread of trachoma.

Chan, in the same issue of this journal, compares the statistics for 1938 and 1939 with those presented by Chi for 1940-1943.

W. ZENTMAYER.

Tumors

RECURRENT JUVENILE PAPILLOMA OF THE CONJUNCTIVA. J. D. WALKER, Am. J. Ophth. **28:** 751 (July) 1945.

Walker reports a case of papilloma of the conjunctiva which recurred repeatedly, eleven operations being performed during five years of observation and treatment. From the literature it is evident that a papilloma may not be an innocent growth, as a number of cases are reported which show that it is capable of malignant degeneration. The tumor is best removed surgically. If roentgen radiation or radium is used, every precaution should be used to protect the lens.

W. ZENTMAYER.

THE MANAGEMENT OF EPIBULBAR MALIGNANCY. WILFRED E. FRY, Pennsylvania M. J. **48:** 694 (April) 1945.

Fry divides epibulbar tumors into the benign, the potentially malignant and the malignant. The benign tumors are the granulomas, epithelial plaques, papillomas, lipomas, hemangiomas, lymphangiomas and dermoids; the potentially malignant tumors are the nevi and malignant papillomas and the malignant tumors are the sarcomas, the epitheliomas and precancerous melanosis.

Two cases are reported, showing, respectively, (1) that sarcoma of the limbus may remain localized at the limbus for a number of years without extensive involvement of the cornea, without intraocular extension and without distant metastasis and (2) that epithelioma of the limbus may extend into the cornea at an early stage.

W. ZENTMAYER.

Vision

VISUAL SYMPTOMS CAUSED BY DIGITALIS. F. D. CARROLL, Am. J. Ophth. **28:** 373 (April) 1945.

This is a report on 6 patients who had visual symptoms due to digitalis. These symptoms consisted in colored vision, chiefly white, green, yellow or red; flashes of light; positive colored scotomas, and other visual hallucinations. There was no change in the visual acuity or the usual fields of these patients, but the literature suggests that if the intoxication is sufficiently profound a temporary cortical type of blindness may result. This condition may occur in patients receiving what is considered a normal dose of the drug and may be the only symptom present. Recovery takes place within two weeks after administration of the digitalis is stopped.

W. S. REESE.

A CONTRIBUTION TO THE THEORY OF BINOCULAR VISION SUPPORTED BY THREE CASES OF LATENT NYSTAGMUS. A. POSNER, Am. J. Ophth. **28:** 392 (April) 1945.

Three cases of latent nystagmus with amblyopia of one eye are presented.

The nystagmus may be interpreted as a modification of normal fixation movements.

The nystagmus is inhibited by a minimal amount of binocular vision, which apparently brings into play a higher degree of cortical control than does monocular vision.

Observations made in these cases confirm the validity of Verhoeff's replacement theory of binocular vision.

The replacement theory also serves to explain the phenomenon of ocular dominance.

W. S. REESE.

STUDIES IN DARK ADAPTATION AS A MEANS OF DETECTING DEFICIENCY OF VITAMIN A. G. W. ROBERTSON and J. YUDKIN, Brit. J. Ophth. **28:** 556 (Nov.) 1944.

In this study, the instrument used was a modified and improved form of the adaptometer described by Hames (*Tr. Ophth. Soc. U. Kingdom* **48:** 103, 1938). In the measurements of final rod threshold, groups of 3 or 4 subjects each enter a dark room directly from their work, without any previous light adaptation. After the subjects have been in the dark for thirty-five or forty minutes, the dark adaptation of each subject is measured by noting the minimal amount of light which can just be seen. The 3 or 4 subjects in each group are tested in turn and then tested a second time in the same order. The second value is taken as the measure of dark-adapting capacity. The following summary is made: Measurements of dark adaptation may be used to assess nutritional level with respect to vitamin A either by determination of the effects of the vitamin on dark adaptation or by comparison of the values for dark adaptation in various groups. The latter method, while not providing such conclusive evidence as the former, may sometimes give presumptive evidence of the relative nutritional status with respect to the vitamin, and for technical reasons it may be the only feasible procedure. Using the former method of measuring vitamin A lability, the authors showed that deficiency of vitamin A existed in a small group of laboratory workers and in a large group of Birmingham (England) factory workers. Of these workers, 147 who took vitamin supplements showed a statistically significant improvement in dark adaptation, while

188 controls showed no significant change. Three groups of school children showed no sign of vitamin A deficiency with this method.

By comparison of the values for dark adaptation in various other groups, it was shown that the Birmingham factory workers and a group of Sheffield factory workers had significantly poorer dark adaptation than the three groups of school children whose dark adaptation had been shown to be unaffected by vitamin A.

W. ZENTMAYER.

Vitreous

RUPTURE OF CHOROID ASSOCIATED WITH CYST OF VITREOUS. E. ROSEN, Brit. J. Ophth. **29:** 486 (Sept.) 1945.

The patient was a Negro aged 21 years. A batted ball struck the summit of the cornea of the right eye apparently directly, as the cornea was abrased. Two months after the injury there was a break in the sphincter of the iris with a segment of atrophy of the iris stroma. There was a choroidal tear in the macular region, and over its center was a biconvex band, the terminals of which were frayed out in a semicircular astral radiation. About 6 disk diameters below the disk was a peculiar cystic structure two and a half times the size of the disk and shaped like an apple. This sphere was loosely suspended from a pedicle and floated on movements of the globe, disclosing a phenomenon of variation in color from yellow to pink. The cyst would settle into a pocket formed by a proliferated connective tissue mass. Examination one month later showed that the cyst had ruptured.

The article is illustrated. W. ZENTMAYER.

Therapeutics

PENICILLIN IN OPHTHALMOLOGY. ARNOLD SORSBY, Brit. M. J. **2:** 542 (Oct. 20) 1945.

Sorsby, on introducing the subject of penicillin therapy at a meeting of the section of ophthalmology of the Royal Society of Medicine, on Oct. 11, 1945, stated that penicillin, as far as the eye was concerned, could not be regarded as a nontoxic substance or as a readily diffusible one. The absence of agreement among observers is partly due to the fact that samples of penicillin available at the moment were largely impure. He stated that it is possible to give penicillin in daily subconjunctival injections if the total dose is not more than 500 or 600 Oxford units. A constant corneal bath produced considerable penetration, and higher concentrations could be reached in the aqueous if ionization was also used; but the combined method was difficult in practice. It has not been found possible experimentally to obtain an adequate concentration of penicillin in the anterior chamber with the use of ointment. If a massive dose was used, slight penetration was obtained, but the difficulty with all these procedures was to obtain an adequate concentration in the eye. With subconjunctival injections a considerable concentration could be reached in the aqueous and vitreous; the drawback was the rapid excretion. After three hours hardly any penicillin was left in the anterior chamber; and, although the excretion was less rapid in the vitreous, the vitreous was extremely intolerant to injections of any type, and the author would not inject more than 200 or 300 units.

There is now a voluminous literature on the local uses of penicillin. The author mentioned 123 cases of ophthalmia neonatorum in which the drug was used during the last year. With a dose of 500 units per cubic centimeter, treatment was successful in 3 of 8 cases; with 1,000 units, in 4 of 7 cases; with 1,500 units, in 5 of 9 cases; and with 2,500 units, in 78 of 98 cases, with relapse in 13 cases and failures in 7 cases. Penicillin used locally was effective over the whole range of micro-organisms occurring with ophthalmia neonatorum. Frequency as well as concentration was important. When penicillin was applied at intervals of one hour, the results were good; at intervals of one-half hour they were better, and at intervals of five minutes, better still. For technical reasons it was not possible to institute the constant drip, and the author and his associates were instilling penicillin at intervals of one minute, and in most cases the condition was under control within half an hour.

Penicillin had been used in a number of ocular conditions. Good control of hypopyon ulcer was obtained with penicillin drops. Trachoma is a condition for which one would not expect the drug to be effective, since it is a virus infection. Sorsby had tried penicillin in 3 cases of trachoma, but only 1 was a fresh case; in this a good result was obtained. In cases of iridocyclitis the results so far reported in the literature have not been significant. The author had used penicillin in 1 case of sympathetic ophthalmia, with no very encouraging result.

The author feels that in penicillin one has a powerful agent but a limited one. It does not readily penetrate into the eye. Its extreme value is in local application for external infections. Unlike the sulfonamide compounds, it is not inactivated by pus, but in treatment of intraocular inflammation neither the sulfonamides nor penicillin has been helpful.

ARNOLD KNAPP.

Society Transactions

EDITED BY DR. W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

THOMAS H. JOHNSON, M.D., *Chairman*

WENDELL L. HUGHES, M.D., *Secretary*

Regular Meeting, May 21, 1945

Ocular Sporotrichosis. DR. DAN M. GORDON, Detroit.

The tenth case of ocular sporotrichosis to be reported in the United States was presented. The patient came to the eye clinic of the New York Hospital with an "ulcerated" area on her left upper eyelid. This was followed by the development of a chain of subcutaneous nodules and associated lymphangitis. Cultures on Sabouraud's medium yielded a rich growth of the Sporotrichum schencki. The patient was placed under oral treatment with potassium iodide and recovered. Pronouncement of cure was based on disappearance of the lesions and a sterile culture.

DISCUSSION

DR. GEORGE M. LEWIS (by invitation): In most cases of sporotrichosis, as Dr. Gordon mentioned, a finger or hand is first affected, and then the lesions extend proximally up the arm, along the lymphatic chain. The initial lesion is often granulomatous and pus producing and may be confused with primary syphilis or pyoderma. When the second lesion appears, the diagnosis of sporotrichosis is not so difficult. In the laboratory diagnosis of this disease one will fail to demonstrate the organism if dependence is placed on stained smears or wet preparations. The diagnosis of sporotrichosis, unlike that of actinomycosis, is made from culture. The organism is seldom demonstrable in the stained preparation or the wet specimen. In Dr. Gordon's case the diagnosis was easily made from culture once the condition was considered; but it is apparent that a certain degree of clinical acuity was needed even for consideration of sporotrichosis in this location.

The rare involvement of the eyelids is of interest. In cases of nail polish dermatitis the eyelids and the side of the neck are the favorite sites. The frequent contact of the hands with the face and the greater sensitivity of the eyelids than other portions of the face account for this occurrence. There may be an analogy between sensitization dermatitis and fungous infection; nevertheless, it seems logical that secondary autoinoculation is more frequently responsible for involvement of the eyelids with this infection.

Dr. Gordon's slides illustrated a case of sporotrichosis appearing at the base of the thumb, with secondary lesions occurring along the lymphatic chain, and another case with involvement of the left lower eyelid and the left cheek. The infection in the second case was acquired by contact while the patient was caring for her daughter, who had the conventional type of sporotrichosis on the arm.

The organism has a typical appearance in culture on dextrose agar. The color is brown to black, and the surface configuration is constant. The growth itself has a pasty consistency and is well circumscribed. To verify the diagnosis, a hanging drop is made from the culture, and pyriform spores are noted in groups on the ends of small lateral branches.

As Dr. Gordon stated, the treatment is usually simple, and satisfactory results may confidently be expected. Potassium iodide should be prescribed in increasing doses. The use of roentgen radiation may sometimes be considered. An occasional patient is intolerant to iodides, and sulfadiazine may then be substituted.

DR. A. RUSSELL SHERMAN, Newark, N. J.: I should like to ask Dr. Gordon whether there is a relation between this condition and infection with Streptothrix or Leptothrix. Do these conditions resemble each other?

DR. FRANK A. VESEY: I should like to ask Dr. Gordon whether any evidence of relative immunity has been established, or whether this condition is equally infectious to every one.

DR. DAN M. GORDON, Detroit: Infection with Streptothrix is usually seen in the conjunctival sac. I believe the only resemblance between Sporothrix and this fungus is the "ix" at the end of the name; the organisms are entirely different. Streptotrichosis is much more common; I saw a great deal of it in Chicago. The leptothrix is the organism found in Parinaud's conjunctivitis.

The question of immunity is an interesting one. I do not believe a single case of the disease in the American Indian has ever been reported. Two investigators have done considerable dermatologic work among the Indians. There was a case of the disease in a half-breed Indian. One case of sporotrichosis was reported by the Harvard expedition to Africa in 1937 or thereabouts; there may have been others, but in a paper published in 1943 only 1 African case was recorded.

I thought some one would ask about the mechanism of iodide therapy. Iodides are not specific in that they destroy the organism; but

they produce a fibroblastic reaction which walls off the organism. Loes stated that before he recognized sporotrichosis he saw a case of a condition which in retrospect he thought was sporotrichosis and which he diagnosed at the time as syphilis. The patient died in two months.

Complications Incident to Simple Intracapsular Extraction: Procedures Designed Toward Prevention. Dr. Frank Bracken.

This paper is published in full in this issue of the Archives, page 430.

Precisional Errors in Measurement of Squint and Phoria. Mr. Otto A. Putnam and Dr. J. V. D. Quereau, Reading, Pa.

This paper was published in full in the July issue of the Archives, page 7.

DISCUSSION

Dr. LeGrand H. Hardy: Several important factors, present singly or in combination, may lead the incautious ophthalmologist into error in measuring squint and heterophoria with prisms.

1. The label on the prism. Most prisms bear a number, but to what unit this number refers is rarely stated. It may refer to the prism diopter (△), the centrad (▽), the deviation produced at the position of minimal deviation or the apex angle of the prism. The unit is usually assumed to to be the prism diopter. However, precise measurements carried out on this assumption over a period of several years on more than twenty sets of prisms showed that many of them were inaccurately labeled, the error at times reaching over 100 per cent. The most serious errors arose when the label referring to the apex angle, in degrees, was mistakenly assumed to refer to the power, in prism diopters.

2. The position in which the prism is held before the eye. The effect depends on the distance from the eye at which the prism is held and its angular rotation about one or more of three axes.

(*a*) A 40 △ prism can accurately correct a 40 △ deviation of the eye only under two conditions: first, when the fixation object is at infinity, and, second, when the effective surface of the prism is at the center of rotation of the eye. Since in measurement of a deviation for near vision the first condition must be violated and in practice the second condition can never be achieved, it follows that a 40 △ prism can never neutralize 40 △ of deviation in making a near point test. This is analogous to the change in effectivity of corrective lenses when placed in front of the eye: The effectivity of a lens will vary with change of its distance from the principal point of the eye.

When one considers the measurement of the deviation of a single eye, it is immediately obvious that the relationship can be expressed thus:

$$D = d + e$$

where D is the power of the prism, *d* is the deviation of the eye from the primary position and *e* is the angle, determined by two factors: (1) the proximity of the fixation point to the eye and (2) the distance at which the prism is held from the eye. The closer the fixation point is to the eye, the greater will be the angle *e*; likewise, the greater the distance between the prism and the center of rotation of the eye, the greater will be the angle *e*.

A simple calculation for a single eye shows that if the deviation, *d*, is 30° (57.7 △) and the fixation distance is 10 inches (25 cm.) the prism necessary to correct this deviation will be 66.4 △ if held 1 inch (2.5 cm.) in front of the eye, or 83 △ if held 3 inches (7.5 cm.) in front of the eye.

When one considers two eyes which alternately fixate a near object, a new factor is introduced. Since the deviation is now measured not from the position of parallelism of the optic axes but from the lines joining the centers of rotations with the fixation point, this new factor will require a reorientation of the prism in convergent and divergent deviations. As one keeps the fixation object in the near field but moves it into the secondary positions of gaze, one introduces more complicating factors, which may disturb analyses unless properly evaluated. It is rare to see the fixation distance carefully maintained in these investigations involving the secondary positions of gaze, and even rarer to see the Prentice position of the prism and its effectivity distance maintained.

Aside from the marked change in effectivity brought about by changing the distance between the eye and the prism, other important positional factors operate.

(*b*) Rotation about the optic axis changes the effect of the prism from base in (or base out) to base up (or base down).

When strong prisms are used, this is an important factor to guard against.

(*c*) Rotation about a frontal horizontal axis from the base horizontal position produces complicated spatial distortion. The optical effects of this type of rotation have been analyzed by Southall.

(*d*) Rotation about a vertical axis from the base horizontal position produces little known but marked changes in power. The use of the term "prism diopter," involving, as it does, the fundamental postulate of Prentice (all power at one surface), introduces serious dangers. It is a valuable and convenient concept to use in prescribing therapeutic prisms to be incorporated in lenses, and its utility is conveniently and safely carried into the field of weak measuring prisms; but when powers above 20 △ are utilized the danger of producing large errors

increases rapidly. A change of almost 20 △ can be effected by wrongly positioning a 1 △ prism. Such an effect probably never occurs since it would require almost 80° of rotation of the prism. However, with a 30 △ prism only 22° of rotation is required to increase its effective power to 87 △; with a 40 △ prism a rotation of 20° will increase its effective power to 97 △, and with a 50 △ prism a rotation of only 8.5° is needed to increase its effective power to almost 104 △.

From these considerations, some practical rules may be formulated:

1. In making near point measurements the prism should be held as close as possible to the eye. This reduces the effectivity factor.

2. The use of a 16 or a 13 inch (40 or 33 cm.) testing distance results in less error, owing to change in effectivity, than the use of a 10 inch (25 cm.) testing distance. This advantage must be evaluated against the concomitant reduction in accommodation.

3. A squint of over 40 △ should never be measured with prisms unless the power is split, approximately half being held before each eye. The error introduced by the inadvertent 10° rotation of two 20 △ prisms amounts to 2×2.75, or 5.5 △, whereas the error introduced by the same amount (10°) of rotation of one 40 △ prism amounts to 17.5 △. (It should be noted that use of the term "prism diopter" is invalid for any position except the Prentice.)

4. In holding prisms before an eye, it is always safer to err by approaching the position of minimal deviation than by turning away from it. Such a maneuver has two advantages: (1) For a given rotation much less power effect is produced, and (2) the effect produced serves to detract from the effectivity factor rather than add to it. Thus, two errors tend to compensate for each other rather than to summate.

This discussion has considered only a few of the most elementary problems involved. Carrying these considerations into measurements involving the secondary positions of gaze has been part of the analytic work which Dr. Quereau and Mr. Putnam have imposed on themselves. I congratulate them and hope they will continue.

DR. JAMES WATSON WHITE: Dr. Quereau and Mr. Putnam have brought up two important points. It is important in making a diagnosis to get the proportionate differences, not only for distance and near vision but in the various direactions of gaze. Whether the error is 3 or 5 prism degrees makes no difference in the diagnosis, as I see it, because the same variance from the true value is carried with a moderate degree of accuracy for distance and for near vision in the cardinal positions and would make no difference in diagnosis or in treatment.

I am afraid that Dr. Hardy's lucid description will scare off many people who do not understand this problem. I believe that if research men would work out these problems in the laboratory and tell the ophthalmologists what to do, and how, more progress would be made and more practical work done.

DR. JOSEPH I. PASCAL: The presentation of Dr. Quereau and Mr. Putnam was very instructive. Persons interested in this work will not find the mathematics hard to follow. But I want to ask Dr. Hardy a question about his objections to the prism diopter as a unit for prism measurement. Since the error introduced is due primarily to the prism not being held properly, that is, wit. the outer surface vertical, what difference would it make if the Jackson method of minimum deviation were used and the prism not held properly? If the prism is held the way Prentice intended it to be held, there is no error. If a prism made on the angle of minimum deviation is not held properly, that is, in the required position, which is symmetrically inclined to patient and examiner, would not similar errors be introduced?

DR. LEGRAND H. HARDY: The answer to Dr. Pascal's question is "no." The position from which a rotational error deviates is not a matter of indifference. From the graph of power plotted against angle of incidence, it is quite clear that the Prentice position of holding occupies a point on the curve at which errors of rotation of slight degree may cause pronounced changes in power. This is not true of the position of minimal deviation. The "split position" lies between the two. For example, a 40 △ prism exhibits 40 △ of effect in the Prentice position. If the angle of incidence is rotated 10° toward the apex, this power effect will reach 57 △. The same prism shows 32 △ of power in the position of minimal deviation (if, for the sake of comparison, one is permitted to use the term "prism diopters" for any position except the Prentice). Rotating the prism 10° toward the apex from the position of minimal deviation will change its effective power from 32 to 33 △. Fortunately, most ophthalmologists do not use the Prentice position of holding. They naively hold the prism in what I have called the "split position," which is nearer the position of minimal deviation than the Prentice position. In the "split position" a 40 △ prism exerts a comparative power of 32.6 △, and a rotation of 10° toward the apex from the position changes the comparative power to about 36 △. This should make it perfectly clear that in using high-powered metric (measuring) prisms the Prentice position is to be avoided, and, I believe, it constitutes a rational explanation of why one almost instinctively avoids the Prentice position, in spite of the fact that one may not, legitimately, use the term "prism diopters" for any but the Prentice position. For the incorpora-

tion of therapeutic prisms (up to 8 △) into lenses small departures from the Prentice position are unimportant, and the ease of using the Prentice method of calculating and numbering prisms justifies its application. For strong metric prisms used in measuring squint these statements do not remain valid.

Dr. White's perturbation should be relieved by the four very practical rules I formulated in the opening discussion.

Aqueous Fibrin Fixation of Corneal Transplants in the Rabbit. Dr. Herbert M. Katzin.

This paper will be published in full in a future issue of the Archives.

Efficiency of the Lens: A Clinical Concept. Dr. Joseph I. Pascal.

The effective power of a plus or minus correcting lens is changed when the lens is moved farther from or nearer the eye. This is known as change in effectivity. Another feature of change in the power of the lens not dependent on movement may be called lens efficiency. The inevitable separation of the correcting lens from the principal plane of the eye imparts to the convex lens positive efficiency because the correcting lens is always less than the true hypermetropic error it corrects in the principal plane of the eye. Conversely, the concave lens has negative efficiency because the concave correcting lens is always more than the true amount of myopia it corrects. Thus, a patient whose error is corrected with a — 30.00 D. lens has a true myopia of only about 20.00 D.

A similar gradation of efficiency may be postulated with respect to the accommodation. Whereas the person with natural emmetropia accommodates 1.00 D. for every diopter of distance, the person with corrected ametropia accommodates either more or less than 1.00 D. for every diopter of distance. If the accommodation of the natural emmetrope is considered as normal efficiency, the lens of the person with corrected hypermetropia has negative efficiency and the lens of the person with corrected myopia has positive efficiency. The reason is that the person with corrected hypermetropia always has to accommodate more than the dioptric value of the object distance, while the person with corrected myopia has to accommodate less than the dioptric value of the object distance.

Fundamentally, this difference in accommodadative efficiency is due to the fact that each person with corrected ametropia has what may be considered his own basic accommodative unit. While the accommodative unit of the natural emmetrope is 1.00 D., the accommodative unit of the person with corrected hypermetropia is always more than 1.00 D. and that of the person with corrected myopia is always less than 1.00 D. Thus, the accommodative unit of the person with corrected hypermetropia of 5.00 D. is 1.23 D. and that of the person with corrected myopia of 5.00 D. is only 0.83 D. (calculation based on 20 mm. as the distance of the lens from the principal plane of the eye).

For a dioptric distance of 4.00 D. (0.25 meter) the natural emmetrope accommodates 4.00 D.; the person with corrected hypermetropia of 5.00 D. accommodates four times his basic unit, which amounts to 4.92 D., and the person with corrected myopia of 5.00 D. accommodates four times his basic unit, which amounts to only 3.32 D. Because of the different accommodative units, the person with corrected myopia becomes presbyopic later than the person with corrected hypermetropia. For the same reason, the same near point of accommodation shows more true accommodative power in the subject with corrected hypermetropia than in the subject with corrected myopia.

In the presbyopic correction of anisometropia, the same addition can never place both eyes in focus for any near distance. The more hypermetropic eye, like the less myopic eye, needs a stronger addition to compensate for the different accommodative units. This difference in units also leads to a variability of astigmatic errors for near distance.

The lens which replaces the accommodation also has positive or negative efficiency, depending on whether its power is less or more, respectively, than the accommodation it replaces. The lens which replaces the accommodation in the person with natural emmetropia has negative efficiency; it is always a little more than the accommodation it replaces, and it increases in power according as it replaces the first, second, third or fourth diopter of accommodation. Thus, it takes a + 1.02 D. lens to replace the first diopter of accommodation, a + 1.06 D. lens to replace the second diopter, a + 1.11 D. lens to replace the third diopter, a + 1.16 D lens to replace the fourth diopter, and so on. The replacing lens for corrected hypermetropia has positive efficiency as long as it replaces part or all of the accommodation that can be used to neutralize the hypermetropia. Thereafter it has negative efficiency. The replacing lens in corrected myopia always has negative efficiency.

Another feature of lens efficiency concerns the form of the correcting lens. Lenses of different form all of which have the same vertex power have the same efficiency for distant, but not for near, vision. For near vision, the double convex lens has the greatest efficiency; the meniscus convex, the least efficiency.

DISCUSSION

Dr. Arthur Linksz, Hanover, N. H.: Any one who is interested in refined refraction must be enthusiastic about Dr. Pascal's presentation. Within the last few years Dr. Hughes and Dr.

Sugar have published papers in which they showed that there are cases in which the axis of astigmatism for near vision may be different from that for distance. About a year ago Dr. Pascal himself added to knowledge in this field, demonstrating that the effectivity of an astigmatic correction is, for intrinsic reasons, different for near and for distant vision. In work on aniseikonia I have been confronted in many cases with the fact that the aniseikonic correction for near vision may be different from the one for distance. All these data remind one that a good deal of basic research is still necessary to refine refraction for close work, and especially the method of addition in close work for presbyopic persons. As Dr. Pascal has just shown, this is not as simple as it is usually thought to be.

One point needs especial emphasis. Dr. Pascal has given a new fundamental concept in what he calls the "accommodative unit." This is a concept with which we all as ophthalmologists will have to become thoroughly familiar. We may hope that much eyestrain, distress and difficulty will be avoided in the future by prescribing corrections for near work in terms of accommodative units and accommodative efficiency rather than in terms of the rigid system which has so far been used.

DR. JOSEPH I. PASCAL: This work is not difficult and can be simplified if one takes the concept of the basic accommodative unit. Each refractive condition, after being corrected, has its own basic accommodative unit. In cases of anisometropia, when the eyes accommodate for any distance — 1 or 2 D. — the difference between the accommodation of the two eyes will be the difference between their accommodative units and the multiples thereof. The same applies to astigmatism, in which each principal meridian has its own accommodative unit. In general, this concept of a basic accommodative unit clarifies, I think, many things which seemed difficult before.

CORRECTION

In the report of the American Ophthalmological Society in the August issue (ARCH. OPHTH. **34**:163, 1945) the following remarks were omitted from the discussion on the paper by Dr. Algernon Reese, entitled "Deep Chamber Glaucoma Due to Formation of a Cuticular Product in the Filtration Angle," (bottom of the first column, continuing the discussions by Dr. Verhoeff):

That bullous keratitis and the related condition, pannus degenerativus, occur in many of the eyes such as Dr. Reese has described is of no significance in regard to the character of the original changes in the ligament but does indicate that the glaucoma was in an advanced stage and had damaged the corneal endothelium. Therefore, while the occurrence of bullous keratitis suggests that alteration of the endothelium led to the formation of elastic membrane in the angle, it also suggests that this formation occurred after the glaucoma had been long established.

If, as Dr. Reese asserts, the conditions that lead to the formation of membrane over the ligament are the same as those that exist in cases of epithelial dystrophy, one would expect that glaucoma would be a frequent complication of this disorder. This, however, is certainly not the case. Also, since warts on Descemet's membrane are commonly found at the periphery of senile eyes, one would expect on the basis of Dr. Reese's views that glaucoma would occur far more frequently in senile eyes than it actually does.

In spite of everything I have said, I am convinced that in Dr. Reese's cases the glaucoma was produced by the changes he describes. I reconcile his observation with mine by assuming that in his cases a tendency to endothelial proliferation existed and that this finally caused obstruction to outflow, and by assuming that in my cases the tendency did not exist but that the changes over the ligament, including the less extensive formation of elastic membrane, resulted from the glaucoma. In other words, I think that Dr. Reese has made a remarkable and highly important discovery.

DR. FREDERICK A. DAVIS, Madison, Wis.: I have had a case recently which may belong to the group described by Dr. Reese.

The patient, a married woman aged 63, was first seen ten weeks ago. She complained of acute pain and loss of vision in her right eye. The pain was excruciating and was accompanied with nausea and vomiting. She had not noticed any reduction in vision prior to this attack, which occurred two weeks before she consulted me. Local treatment and administration of sedatives by her family physician had brought slight relief, but vision had grown progressively worse. Examination revealed a moderately inflamed globe. The cornea was steamy, having a ground-glass appearance. The anterior chamber was deep. The aqueous appeared to be clear and was free of cells so far as could be determined with the slit lamp. No keratitic precipitates were present. The pupil was dilated and fixed, measuring 4 to 5 mm. No definite synechias could be seen; the iris was atrophic. Tension was 59 mm. in the right eye and 17 mm. in the left eye, with the Schiøtz tonometer. Vision was 20/70 in the right eye and 20/20 in the left eye.

A whitish mass of some foreign substance was present in the temporal and inferior region of the chamber angle. It appeared to be fused with the cornea. It did not present the ordinary characteristics of an inflammatory exudate, nor did it appear to be a new growth.

The fundus could be seen indistinctly, but there appeared to be moderate temporal cupping of the disk. Gonioscopic studies were not made because of the corneal haze and inflammation.

The patient was treated intensively in the hospital for nineteen days with miotics and hot applications.

The acute inflammatory symptoms subsided somewhat, and the tension was reduced to 37 mm. Schiøtz. An Elliott trephine operation was performed by my assistant, Dr. P. A. Duehr, with very satisfactory results. The tension fell to 17 mm.; the corneal edema disappeared, and the eye gradually became quiet. Study of the fundus revealed typical glaucomatous cupping of the disk. The field of vision showed concentric contraction to about 20 degrees. The pupil has remained semidilated and fixed at 5 mm. No synechias can be seen. The iris is atrophic. Vision is now 20/50.

The left eye is apparently unaffected, fields, fundus and tension being normal.

The patient is still under observation, and further studies of the chamber angle will be carried out.

Dr. Algernon Reese, New York: Dr. Kronfeld points out the importance which central vision and visual fields would have been in establishing whether or not these patients came to enucleation early or whether or not the cases on which this report is based was cases of far advanced glaucoma. The corneal changes were so extensive that central vision or the visual fields would not be an accurate criterion. Also, the corneal changes prevented an accurate view of the interior of the eye.

Dr. Verhoeff points out that this type of glaucoma should be more prevalent in older people. The average age in these cases was only 50 years. He also states that glaucoma should be more prevalent in cases of cornea guttata. I agree with him in both these contentions. Furthermore, one would expect glaucoma due to this process to be present in both eyes.

I believe the case which Dr. Davis cites belongs to this group. Dr. Friedenwald poses the question whether glaucoma with an abnormally deep anterior chamber is due to this type of lesion in all cases. In my series, 26 cases were thought to belong to this group, but there were 16 additional cases of deep chamber glaucoma with open angle which did not belong here.

I very much appreciate the discussion, and I think Dr. Verhoeff has been particularly generous.

Correspondence

REQUEST FOR COPIES OF THE ARCHIVES OF OPHTHALMOLOGY FOR OPHTHALMOLOGISTS ABROAD

To the Editor:—Since 1941 American publications have not been available to most of the Continental centers and investigators. I have had several letters from ophthalmologists abroad indicating the desire and need for ophthalmic literature which has appeared during these war years.

It occurred to me that some subscribers of the Archives with copies for the past several years would be willing to send their copies abroad to ophthalmic centers or to individuals. If such subscribers will write to the Howe Library of Ophthalmology, c/o Miss Jeanette Loessel, 243 Charles Street, Boston 14, a name and address will be provided to which the Archives may be sent. Arrangements are being made to reimburse the donors for the cost of mailing.

David G. Cogan, M.D., Boston.
243 Charles Street.

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All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. K. S. Sun.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, China.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

Faculty of Ophthalmologists

President: Brig. Sir Stewart Duke-Elder, 63 Harley St., London, W. 1, England.
Secretary: Mr. Frank W. Law, 45 Lincoln's Inn Fields, London, W. C. 2., England.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Imre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Mr. John Foster, 45 Park Sq., Leeds.
Secretary: Mr. William M. Muirhead, 70 Upper Hanover St., Sheffield.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. J. Ringland Anderson, Astor House, 108 Collins St., Melbourne, Victoria.
Secretary: Dr. D. A. Williams, 27 Commonwealth St., Sydney.
Place: Melbourne. Time: Oct. 20-26, 1946.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secetary, Dr. Mohammed Khalil.

Ophthalmological Society of Hospital de Nuestra Señora de la Luz

Chairman: Dr. Manuel J. Icaza y Dublán, México, D. F., Mexico.
Secretary: Dr. Jorge Meyrán, México, D. F., Mexico.

Ophthalmological Society of South Africa

President: Dr. A. W. Sichel, National Mutual Bldg., Church Square, Cape Town.
Secretary: Dr. J. K. de Kock, Groote Kerk Bldg., 32 Parliament St., Cape Town.

Ophthalmological Society of the United Kingdom

President: Mr. Charles B. Goulden, 89 Harley St., London.
Secretary: Mr. Frank W. Law, 30 Devonshire Pl., London, W. 1.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 4-6, 1946.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

SÃO PAULO SOCIETY OF OPHTHALMOLOGY

President: Silvio de Almeida Toledo, Barão de Ilapetininga St., 88, 5º Andar, São Paulo, Brazil.
Secretary: Dr. Plinio de Toledo Piza, Enfermaria Santo Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. S. Spence Meighan, 13 Woodside Pl., Glasgow, C. 3.
Secretary: Dr. Alexander Garrow, 15 Woodside Pl., Glasgow, C. 3.
Place: Edinburgh and Glasgow, in rotation.

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Arturo Etchemendigaray, Villa Constitución, Santa Fé.
Place: Rosario. Time: Last Saturday of every month, April to November. All correspondence should be addressed to the President.

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS

President: Prof. Hilton Rocha, Rua Rio de Janeiro 2251, Bello Horizonte, Minas Geraes, Brazil.
Secretary: Dr. Ennio Coscarelli, Rua Aimorés 1697, Bello Horizonte, Minas Geraes, Brazil.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DE RIO GRANDE DO SUL

President: Dr. Luiz Assumpção Osorio, Edificio Vera Cruz, Apartamento 134, Porto Alegre, Rio Grande do Sul.
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul.

SOCIEDADE DE OPHTHALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4, III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Conrad Berens, 35 E. 70th St., New York.
Secretary-Treasurer: Major Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.
Assistant Secretary-Treasurer: Dr. Hunter Romaine, 35 E. 70th St., New York.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

Canadian Ophthalmological Society

President: Walter W. Wright, 170 St. George St., Toronto 5.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

National Society for the Prevention of Blindness

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear Nose and Throat

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Central Illinois Society of Ophthalmology and Otolaryngology

President: Dr. Watson Gailey, 1000 N. Main St., Bloomington, Ill.
Secretary-Treasurer: Dr. William F. Hubble, 861-867 Citizens Bldg., Decatur, Ill.

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. Theodore L. Terry, 140 Marlborough St., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. James H. Mathews, 1317 Marion St., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. L. D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Harold H. Heuser, 207 Davidson Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July, August and September.

Sioux Valley Eye and Ear Academy

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

Southwestern Academy of Eye, Ear, Nose and Throat

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

Southwestern Michigan Triological Society

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. Ray Parker, 218 Franklin St., Johnstown, Pa.
Secretary-Treasurer: Dr. J. McClure, Tyson, Deposit Nat'l Bank Bldg., Dubois.

STATE

Arkansas State Medical Society, Eye, Ear, Nose and Throat Section

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

Colorado Ophthalmological Society

President: Dr. C. A. Ringle, 912–9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 526 Walton St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Kansas State Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. W. D. Pittman, Pratt.
Secretary: Dr. Louis R. Haas, 902 N. Broadway, Pittsburg.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Val. H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Medical Society of the State of Pennsylvania, Section on Eye, Ear, Nose and Throat Diseases

Chairman: Dr. Karl M. Houser, 2035 Delancey St., Philadelphia 3.
Secretary: Dr. William T. Hunt Jr., 1205 Spruce St., Philadelphia 7.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Edmond L. Cooper, 1553 Woodward Ave., Detroit 26.
Secretary: Dr. Ralph H. Gilbert, 110 Fulton St. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Karl C. Wold, 1051 Lowry Bldg., St. Paul 2.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul 2.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N. St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. George P. Meyer, 410 Haddon Ave., Camden.
Secretary: Dr. John P. Brennan, 429 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Harold J. Joy, 504 State Tower Bldg., Syracuse 2.
Secretary: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 104 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. L. Diven, City National Bank Bldg., Bismarck.
Secretary-Treasurer: Dr. A. E. Spear, 20 W. Villard, Dickenson.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Lewis T. Buckman, 83 S. Franklin St., Wilkes-Barre.
Secretary Pro Tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society, Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. E. B. Fairbanks, 315 Medical Arts Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Dean Spear, 516 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn 11.
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Ave., Brooklyn 13.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. William H. Howard, 389 Linwood Ave., Buffalo 9.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo 9.
Time: Second Thursday of each month from October to May.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Samuel J. Meyer, 58 E. Washington St., Chicago 2.
Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago 2.
Place: Continental Hotel, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. H. H. Wygand, Guardian Bldg., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Erwin W. Troutman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. T. Rees Williams, 380 E. Town St., Columbus 15, Ohio.
Place: University Club. Time: 6:15 p. m., first Monday of each month, from October to May, inclusive.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. C. B. Collins, 704 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. L. W. O. Janssen, 710 Medical Professional Bldg., Corpus Christi, Texas.
Time: 6:30 p. m., third Tuesday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Ruby K. Daniel, Medical Arts Bldg., Dallas 1, Texas.
Secretary: Dr. Tom Barr, Medical Arts Bldg., Dallas 1, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Wesley G. Reid, 667 Fisher Bldg., Detroit 2.
Place: Club rooms of Wayne County Medical Society.
Time: First Monday of each month, November to April, inclusive.

Detroit Ophthalmological Society

President: Dr. Raymond S. Goux, 545 David Whitney Bldg., Detroit 26.
Secretary: Dr. Arthur Hale, 1609 Eaton Tower, Detroit 26.
Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Lyle J. Logue, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. John T. Stough, 803 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmoolgical and Oto-laryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Francis Carl Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Robert G. Thornburgh, 117 E. 8th St., Long Beach, Calif.
Place: Seaside Hospital. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Secretary-Treasurer: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach 2, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Frank G. Treskow, 411 E. Mason St., Milwaukee 2.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montreal Ophthalmological Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6.30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashville, Tenn.
Place: James Robertson Hotel. Time 6:30 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.
Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Oto-laryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Secretary: Dr. Truman L. Boyes, 654 Madison Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Maurice L. Wieselthier, 1322 Union St., Brooklyn.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York 28.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.
Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. A. Steinberg, 1502 Farnam St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha 2.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Clarence F. Bernatz, Park Bldg., Pittsburgh.
Secretary: Dr. Robert J. Billings, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July. August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. Isaac B. High, 326 N. 5th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from September to July.

Richmond Eye, Ear, Nose and Throat Society

President: Dr. Luther C. Brawner, Professional Bldg., Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. Vincent Jones, 634 N. Grand Blvd., St. Louis.
Secretary: Dr. T. E. Sanders, 508 N. Grand Blvd., St. Louis 3.
Place: Oscar Johnson Institute. Time: Fourth Friday of each month from October to April, inclusive, except December, at 8:00 p. m.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.
Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.
Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.
Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Clarence A. Veasey, 421 W. Riverside Ave., Spokane, Wash.
Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. L. C. Ravin, 316 Michigan St., Toledo 2, Ohio.
Secretary: Dr. W. W. Randolph, 1838 Parkwood Ave., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Harold M. Downey, 1740 M St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Richard W. Wilkinson, 1408 L St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.

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